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RELATION OF THE SYMPATHETIC NERVOUS SYSTEM TO DISEASES OF THE EYE

WITH REGARD TO SURGICAL PROCEDURES

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The sympathetic nervous system embraces the system that may be actuated by external stimuli, such as light (causing reaction of the iris), cutaneous irritation and emotions; the sympathetic portion proper, and the purely autonomic system, sometimes referred to as the parasympathetic system (fig. 1).

Starling [1] described the basic physiology and anatomy as follows:

> All the vasoconstrictor nerves of the human body leave the spinal cord by the anterior roots of the spinal nerves from the first dorsal to the fourth lumbar inclusive. From the roots they pass by the white rami communicantes to the ganglia of the sympathetic chain lying along the front of the vertebral column. Here they take different courses according to their destination. . . . The fibers to the head and neck leave by the first four thoracic nerves, pass into the sympathetic chain through the first dorsal ganglion and ansa Vieusseni to the inferior cervical ganglion, and up the cervical sympathetic trunk to the superior cervical ganglion. Here they end, and the impulses are carried by a fresh relay of fibres, which start from cells in this ganglion and travel as non-medullated fibres on the walls of the carotid artery and its branches.

The internal carotid plexus, an extension from the carotid artery plexus, passes with the internal carotid artery to the cavernous sinus, where it forms the cavernous plexus. From this plexus branches are given off which go to the oculomotor and trochlear nerves and to the ophthalmic division of the trigeminal nerve; it also communicates with the ciliary ganglion through its long root.

The inferior cervical sympathetic ganglion is blended directly with the first thoracic ganglion or connected with it by a short stout portion of the cervical sympathetic trunk. The branches to the vertebral artery are large and they unite with similar branches from the first thoracic ganglion to form a plexus, the vertebral plexus, which accompanies the artery into the posterior fossa of the cranium, where it is con-

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1. Starling, E. H.: Principles of Human Physiology, ed. 5, Philadelphia, Lea & Febiger, 1930, p. 795.

tinued on the basal artery.[2] From this plexus communicating branches pass to the sympathetic plexus on the internal carotid artery and to the cavernous sinus plexus.

In order to cut off all possible sympathetic nerve supply to the eye the inferior cervical sympathetic and the first thoracic ganglion, together with the beginning of the carotid plexus, must be removed. While the removal of the superior cervical sympathetic ganglion would include the greater part of the sympathetic nerve supply to the eye, it would not include the supply via the vertebral sympathetic nerve plexus.

The opinion was stated by Beattie [3] and Lyle [4] that the pupillodilator fibers do not cross in the medulla.

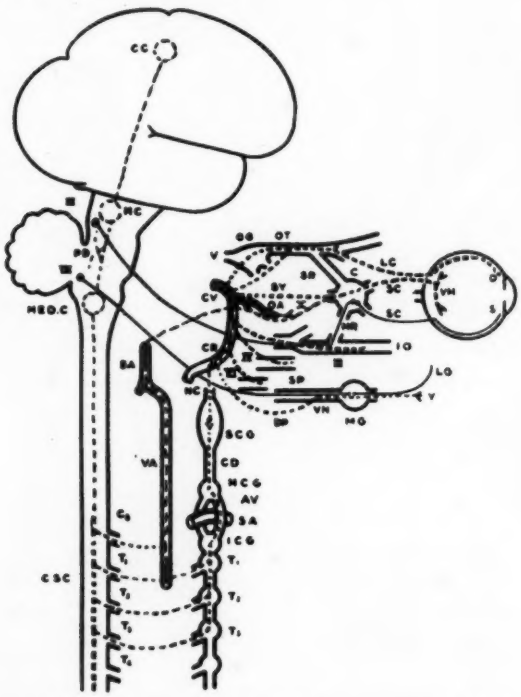

Fig. 1.—Diagram of the sympathetic and parasympathetic systems associated with the eye (from Duke-Elder's "Text-Book of Ophthalmology"). The sympathetic fibers are indicated by broken lines, the parasympathetic fibers by unbroken lines. The letters have the following significance: *AV*, ansa Vieussens; *BA*, basilar artery; *C*, ciliary ganglion; *CD*, cervical part of the spinal cord; *CR*, carotoid plexus; *CSC*, ciliospinal center; *CV*, cavernous plexus; *D*, fibers to the dilator pupillae; *GG*, gasserian ganglion; *ICG*, inferior cervical ganglion; *MCG*, middle cervical ganglion; *MR*, motor root of the ciliary ganglion; *OA*, ophthalmic artery; *S*, fibers to the sphincter pupillae; *SC*, short ciliary nerves; *SCG*, superior cervical ganglion; *T* to *T*, thoracic nerves 1 to 4; *VA*, vertebral artery; *VM*, vasomotor nerves to the eyeball.

2. Morris, H.: Human Anatomy, edited by C. M. Jackson, ed. 4, Philadelphia, P. Blakiston's Son & Co., 1907, pt. 3, p. 1007.

3. Beattie, J.; Duel, A. B., and Ballance, C.: J. Anat. **66**:283 (April) 1932.

4. Lyle, D. J.: Ohio State M. J. **28**:106 (Feb.) 1932.

The object of operation on the sympathetic nervous system is the removal of the vasoconstrictor action in the following diseases affecting the eye, in the interest of improving the nutrition of the eye: atrophy of the optic nerve and retina, chorioretinitis and retinitis pigmentosa. The idea of sympathectomy in case of glaucoma originated in the discovery of the fact that paralysis of the sympathetic nervous system was followed by reduction in ocular tension. Evident disturbance of the sympathetic nervous system occurring in cases of exophthalmic goiter led to the resort to sympathectomy in the search for a cure in this disease.

ATROPHY OF THE OPTIC NERVE

The late Dr. James M. Ball [5] wrote:

> Excision of the cervical portion of the sympathetic nervous system for the relief of optic nerve atrophy was proposed and executed by the writer in 1899. I was led to make this experimental operation for several reasons: (1) The use of glonoin is often followed by an improvement in vision in cases of simple atrophy of the optic nerve. (2) Glonoin enlarges the retinal vessels as has been proved by retinoscopic examination. (3) There is no question that in glaucoma simplex improvement in vision follows sympathectomy. (4) Excision of the cervical sympathetic is followed by an increase in the blood supply of the orbital contents.

Ball's cases [6] are, briefly, as follows:

> CASE 1.—A man 46 years of age had no history of systemic disease. Vision of the right eye was perception of light; vision in the left eye was nil. The diagnosis was atrophy of the optic nerve and retina.
>
> Operation—removal of the right superior cervical ganglion—was performed. Two weeks later there was no improvement in vision. A cilioretinal artery in the upper part of the right disk had doubled in caliber.
>
> CASE 2.—A man 40 years of age had been treated by several ophthalmologists for simple atrophy of the optic nerve, without result. Vision of the right eye was not given; vision of the left eye was perception of fingers at 2 feet (60.9 cm.).
>
> The superior cervical sympathetic ganglion on the left side was removed. Vision of the left eye increased to 20/100. The patient was lost sight of, but one year later he reported that his vision was excellent and that he had resumed his vocation.
>
> CASE 3.—A man 64 years of age had syphilis, for which he was treated for two months. Vision of the right eye began to fail in 1898, and that of the left eye, in 1900.
>
> He was admitted to St. Louis City Hospital on Jan. 29, 1901, and given large doses of mercury and the iodides. The general health was good. Vision in the right eye was nil; vision in the left eye was 6/200. The fundi showed the typical picture of atrophy of the optic nerve. On April 11 the left cervical sympathetic

5. Ball, J. M., in The Relation of the Cervical Sympathetic to the Eye: Papers Read Before Section on Ophthalmology of American Medical Association at Annual Session, New Orleans, La., 1903, Chicago, American Medical Association Press, 1904, p. 91; J. A. M. A. **42**:287 (Jan. 30) 1904.

6. Ball, J. M.: Compt. rend. Cong. internat. d'opht. d'Utrecht, 1900, p. 550.

ganglion was removed. Two days later vision of the left eye was 15/200; on the sixth postoperative day vision of the left eye was 12/200. At the end of two weeks the vision was as before the operation.

Ball reported further:

Suker performed a bilateral sympathectomy in a case of bilateral simple atrophy of the optic nerve. Before the operation the visual acuity was reduced to form perception at two feet. After operation the hand could be recognized at six feet. Two years later the visual acuity was retained.[7]

Ball also reported that Renaud[8] in January 1900 did a bilateral sympathectomy for unilateral atrophy of the optic nerve which followed an attack of optic neuritis existing four months previously. Vision was 6/200. Four months later vision was 20/100.

In Renaud's other case, which was one of simple atrophy, the patient showed in two months improvement in vision from 20/200 to 20/50. Renaud operated on another patient, without success.

Dr. E. Gruening[9] reported a case as follows:

A man 33 years of age had contracted syphilis fifteen years previously. When I saw him in 1902 he was entirely blind as regards his left eye, but saw well centrally with his right; here the field was telescopic, measuring 10 degrees in diameter. A diagnosis of postsyphilitic tabes with atrophy of the optic nerves was made. The right superior cervical sympathetic ganglion was removed. Vision became worse, and in three months the man was totally blind.

In these eight cases of sympathectomy in the treatment of atrophy of the optic nerve there were four failures, and in four patients the condition was reported to have been improved. Ball operated by removing the superior cervical ganglion of the sympathetic nervous system on the side of the eye the vision of which he wished to improve, as did all the surgeons that were cited by Ball.

In June 1934, on the recommendation of A. Löwenstein,[10] the surgical clinic at the University at Prague began to perform periarterial sympathectomy at the division of the carotid artery on patients with ocular disease. The operations were followed by fluctuations of the general blood pressure and of the intra-ocular pressure and of the caliber of the retinal arteries.

Magitot has reported four cases of atrophy of the optic nerve and two of chorioretinitis, in each of which the operation was followed by definite improvement of vision and of the visual fields.

In eight cases of tabetic atrophy of the optic nerve no improvement could be noted.

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7. Suker: New York M. J. **71**:266 (Feb. 24) 1900; cited by Ball.[5]
8. Renaud, E. C.: St. Louis M. Rev. **45**:74, 1902.
9. Gruening: Personal communication to the author.
10. Löwenstein, A.: Arch. f. Ophth. **133**:636 (April) 1935.

EXOPHTHALMIC GOITER

Operations on the cervical portion of the sympathetic nervous system in the treatment of exophthalmic goiter was practiced extensively in the early part of this century. Ball [5] in writing on the subject divided the cases of exophthalmic goiter into three groups:

1st. Those dependent on changes in the central nervous system, in which no operative measure can be of any benefit.

2nd. Those due to disease of the cervical sympathetic in which excision of the cervical sympathetic ganglia may give relief.

3rd. Those arising from excessive thyroid secretion in which thyroidectomy may cure.

The following paragraphs give an abstract of Ball's article:

Jaboulay [11] performed bilateral section of the sympathetic trunk in six cases and unilateral operations in two cases. He recorded two cures, five cases in which the condition improved and one case in which death occurred eighteen months after the operation. In all the cases the exophthalmos was influenced favorably after the first day.

Balacesu [12] removed the superior and middle cervical ganglions with the intervening strand. He reported twenty-seven cases, which were observed for periods varying from one to four years. In all these cases there was rapid and pronounced improvement. In this series of twenty-seven cases there were reported nine cases with cures, eleven cases in which improvement occurred, two cases in which there was no change and five cases in which death occurred, in none of which this could be attributed to the operation.

Total bilateral resection of the cervical sympathetic ganglion is the procedure of choice among the operations for sympathectomy. Of nineteen cases which were reported by Balacesu as occurring in the practice of Jonnesco, Soulié, Faure, Peugnecz and Dépage, in fourteen on the second day after operation the symptoms of exophthalmic goiter disappeared one after another. This improvement continued daily until the cure was complete.

Up to the present time no deaths have been directly attributed to the total excision of the cervical sympathetic ganglion. The results are: cure in 63.8 per cent of the cases, improvement in 18.1 per cent, failure in 18.1 per cent and death in none.

In concluding his article Ball [5] wrote:

In exophthalmic goitre complete excision of the cervical sympathetic is followed by a larger percentage of cures than is any other procedure. Thus far no deaths have occurred. The number of operations, however, is small and final conclusions can be announced only after a large number of cases have been treated by this method.

11. Jaboulay: Chirurgie du grande sympathetique et du corps thyroïde, Paris, O. Doin, 1900, p. 79.

12. Balacesu: Arch. f. klin. Chir. **67**:59, 1902.

Curtis reported seven cases in which sympathectomy was performed for exophthalmic goiter. Of these patients, three died, two from acute hyperthyroidism and one from the effects of the anesthetic. Of the four patients who survived the operation, one had a relapse within nine months and died ultimately of acute endocarditis. One patient showed great improvement at the end of a year and died ultimately of nephritis. One was completely cured at the end of five years, and one showed improvement. Curtis abandoned sympathectomy for partial thyroidectomy when operation was advisable.

At present sympathectomy is not employed in the treatment of exophthalmic goiter, partial thyroidectomy being preferred.

GLAUCOMA

De Schweinitz [13] stated:

> As long ago as the time of Pourfour-du-Petit, that is in 1727, it was observed that after section of the cervical sympathetic the eye was softer and this fact was afterward verified by Claude Bernard and other experimenters. . . .
>
> Abadie, believing that the symptoms of glaucoma can be explained by an excitation of the vaso dilator fibers of the ocular blood vessels, suggested, in 1898, that relief might be obtained by section of the sympathetic in the neck. Soon afterward the first operation of resection of the cervical sympathetic was performed by Jonnesco.

Jonnesco's operation was excision of the superior ganglion of the cervical sympathetic.[14]

In the spring of 1902 I [15] presented the histories of four patients, from two of whom the superior cervical ganglion had been removed from both sides and on two of whom a similar operation had been performed on one side. In all the cases the condition was simple chronic glaucoma. In all Horner's syndrome followed the operation on the side on which sympathectomy had been performed, and in all there was a moderate decrease in the tension of the corresponding eye. In one case a severe attack of glaucoma was experienced about two months after the sympathectomy, the attack occurring on the side on which sympathectomy had been performed in an eye that was almost blind. In one case the more affected eye presented symptoms of more or less acute disturbance, rendering iridectomy advisable. In the remaining cases the eye retained improvement in vision and in the field, but the tension became about as before operation within a few weeks.

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13. de Schweinitz, George E., in The Relation of the Cervical Sympathetic to the Eye: Papers Read Before Section on Ophthalmology of American Medical Association at Annual Session, New Orleans, La., 1903, Chicago, American Medical Association Press, 1904, p. 19; J. A. M. A. **42**:286 (Jan. 30) 1904.

14. Jonnesco, T., and Floresco, N.: Bull. Acad. de méd., Paris **48**:7, 1902.

15. Weeks, John E.: Tr. Am. Ophth. Soc. **9**:441, 1902.

In an article by Dr. William H. Wilder [16] the author assembled the cases reported in the literature to that time, which appear in the table. Wilder stated:

> Myosis, a phase of Horner's syndrome, is mentioned as occurring 40 times and this condition remained for periods ranging from two days to three years; in most cases it seems to be permanent. Conjunctival congestion was noted in 19 cases lasting for a variable time, usually not longer than a few days. Congestion of the side of the face operated on was observed in six cases, in one case lasting three months.

Longer observation of the patients operated on has convinced ophthalmic surgeons that operations on the sympathetic nervous system for relief in glaucoma have produced few lasting, satisfactory results, and the operation has been abandoned for more efficient procedures.

RETINITIS PIGMENTOSA

In two articles N. D. Royle,[17] of Sydney, Australia, has reported the results of operations on the sympathetic nervous system in the treatment of retinitis pigmentosa. Royle apparently favors the operation at the location of the first thoracic sympathetic ganglion. He wrote:

Results of Sympathectomy for Glaucoma in Cases Reported in the Literature

Form of Glaucoma	Number of Cases	Condition Improved	Condition Stationary	Condition Worse
Simple chronic	81	55	23	2
Chronic inflammatory	50	30	18	1
Subacute	18	10	6	2
Acute	12	6	6	0
Absolute	7	2	5	0
Hemorrhagic	7	7	0	0
Buphthalmos	7	4	2	1
Total	182	114	62	6

> If it is desirable to take out the whole of the sympathetic supply to the brain, the branches of the first thoracic ganglion are divided and the ganglion removed; it is probably better to do so in patients suffering from this disease [retinitis pigmentosa]. Operation usually on one side. The usual local symptoms (Horner's syndrome) were present after the operation.

Royle reported operations on fourteen patients. He said:

> Nine, who had come from the Blind Institution, had lost their central fixation . . . in none of these was any permanent improvement obtained. Of the remaining five patients . . . four have maintained improvement and one has lost ground after immediate response.

16. Wilder, William H., in The Relation of the Cervical Sympathetic to the Eye: Papers Read Before Section on Ophthalmology of American Medical Association at Annual Session, New Orleans, La., 1903, Chicago, American Medical Association Press, 1904, p. 43; J. A. M. A. **42**:350 (Feb. 6); 425 (Feb. 13) 1904.

17. Royle, N. D.: (*a*) M. J. Australia **2**:364 (Sept. 13) 1930; (*b*) **2**:111 (July 23) 1932.

In his second case [17b] the patient was a deaf-mute 18 years of age. Vision was 6/6 in each eye. The fields of vision were between 20 and 30 degrees in area when the patient was first seen, on July 6, 1930. The report reads:

> On July 25, 1930, after operation on the right side, the fields were doubled in area. On Jan. 22, 1931, right and left vision was 6/6, and the fields were further enlarged. On Dec. 14, 1931, after operation on the left side, the right and left vision was 6/6; the fields were almost normal. The patient regained his hearing.

Royle concluded as follows:

1. Thoracic sympathetic trunk section affords relief in the treatment of blindness associated with *retinitis pigmentosa.*
2. The relief is more marked in younger patients.
3. Complete blindness of long standing is a contraindication to operation.

Dr. Glen Campbell [18] reported one case, that of a woman aged 24 years. Vision of each eye was 6/12. The pupils were each 6 mm. in diameter and reacted to light. The Wassermann reaction was negative. On Nov. 20, 1930, removal of the superior cervical sympathetic ganglion on the left side was performed. This was followed by Horner's syndrome on that side. Enophthalmos of 4 mm. was found on March 30, 1931; macular vision was the same as before the operation. There was some improvement in the fields of vision on both sides. On October 27 the superior cervical ganglion was removed from the right side. No further improvement was noted. There was no miosis or pseudoptosis on the right side such as occurred on the left side.

Dr. H. H. Kerr [19] reported operations on eight patients with retinitis pigmentosa by bilateral excision of the superior cervical sympathetic ganglion, with immediate improvement in three cases and no improvement in five. In the cases in which improvement occurred the condition was not far advanced. Kerr concluded:

> We believe that operation is definitely contraindicated in cases in which central vision is reduced to less than 10 per cent of normal and where the fields occupy a circle of less than 10 degrees radius.

MacDonald and McKenzie [20] reported four cases in which they operated by sympathectomy. In the first case section of the sympathetic trunk below the second thoracic ganglion was performed, with no improvement. In the second case the right stellate and the second thoracic ganglion were removed, with no improvement. In the third

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18. Campbell, Glen: Canad. M. A. J. **26**:674, 1932.
19. Kerr, H. H.: Am. J. Surg. **28**:364, 1935.
20. MacDonald, A. E., and McKenzie, K. G.: Sympathectomy for Retinitis Pigmentosa, Arch. Ophth. **13**:362 (March) 1935.

case, that of a man 35 years of age, the right stellate ganglion was removed; this was followed by slight enlargement of the field of vision on that side, with no change in the field on the left side. Ten months later the left stellate and second thoracic ganglions extending onto the carotid plexus were removed. The patient stated that he noticed no change in vision. The fields of vision were apparently slightly enlarged. In the fourth case a similar operation was performed on the right side. Six months later there was no change in vision. The final results were very slight improvement in two cases and no improvement in two. The improvement "was more marked in the patient with the shortest history of retinitis pigmentosa."

Walsh and Sloan [21] have presented a comprehensive review of the cases of retinitis pigmentosa reported in the literature in which operations on the sympathetic nervous system have been performed to date, to which three carefully studied cases of their own were added. In their first case, the first and second thoracic ganglions and the superior cervical ganglion on the right side were removed. Horner's syndrome resulted. "Subsequent examination failed to show improvement in vision, visual fields or light sense, or visible changes in the caliber of the retinal vessels."

In their second case the stellate and the superior cervical ganglions on the right side and the superior cervical ganglion on the left side were removed. One month later vision had become reduced from 20/50 to 20/70 in the right eye and from 20/40 to 20/50 in the left. Horner's syndrome was present on both sides.

Their third case was that of a woman aged 35 years. The right stellate and the right superior cervical ganglion and the cervical chain were removed. This was followed by Horner's syndrome on that side. Fourteen months later Horner's syndrome was unchanged. Improvement in the fields of both eyes followed, reaching a maximum in four months, when evidence of regression occurred, but at the end of fourteen months some improvement in the fields was retained. Vision before the operation was 20/70 in the right eye and 20/30 in the left. Fourteen months later vision of the right eye was 20/100 and that of the left was 20/40 plus 1.

Walsh and Sloan concluded:

> The summary of cases shows that unquestionable improvement is infrequent. Ophthalmoscopic examination fails to show increased size of the retinal arteries. The "low" operation may cause vasomotor changes, which in the upper extremity may constitute a disability.

21. Walsh, F. B., and Sloan, L. L.: Results of Cervical Sympathectomy in Pigmentary Degeneration of the Retina, Arch. Ophth. **14**:699 (Nov.) 1935.

De Takáts and Gifford [22] in writing on this subject reviewed the literature and added six cases of their own. They stated: "We have performed altogether eleven sympathectomies on six patients. Of these four were cervicodorsal and seven were superior cervical sympathectomies." In the latter operation the contribution to the eye from the plexus on the vertebral artery carried forward on the basal artery in the cranium is not interrupted.

In case 1 sympathectomy was performed on the left side on Oct. 2, 1933, without producing Horner's syndrome. On Jan. 7, 1934, sympathectomy was performed on the left side; this operation was followed by the appearance of Horner's syndrome on the left side.

In case 4 Horner's syndrome followed cervical sympathectomy on the right side. A similar operation performed on the left side about two months later was not followed by Horner's syndrome. "On the left side, however, no complete sympathetic denervation was accomplished."

In case 5 sympathectomy on the right side was not followed by Horner's syndrome, but sympathectomy on the left side, which was performed later, was followed by Horner's syndrome on that side.

Does the absence of Horner's syndrome after sympathectomy indicate incomplete denervation on the side on which the operation has been performed?

In case 6 cervical sympathectomy was performed on the left side, resulting in incomplete Horner's syndrome. Six months later a similar operation was performed on the right side; this was followed by a complete Horner syndrome. Vision and the visual fields were unchanged. The retinal vessels on the right side were considered to be somewhat larger than those on the left but were quite small on both sides.

In their summary de Takáts and Gifford wrote: "Of eleven operations, four consisted of cervico-dorsal and seven of superior cervical ganglionectomies."

These authors favor the latter operation. They stated:

> The test of complete sympathetic denervation was the failure of cocaine to dilate the contracted pupil. Not one of these patients showed an increase in visual acuity or in visual fields. . . . None of them showed progress of the disease, but the longest period of observation was only a year and a half.

In the thirty-six cases here collected, the pathologic condition apparently was not made worse by the operative procedure in any. Very slight improvement was noted in eleven cases. None of the cases at the time of reporting had been under observation more than two years, and a number had been observed less than two months. The favorable

22. de Takáts, G., and Gifford, S. R.: Cervical Sympathectomy in Retinitis Pigmentosa, Arch. Ophth. **14**:441 (Sept.) 1935.

results were obtained in the younger patients and in those in whom the disease was in the early stage. It is possible that operation on the sympathetic nervous system may serve to retard the development of retinitis pigmentosa or, in some cases, to arrest it when performed at an early stage of the condition.

In regard to the effect of sympathectomy on the size of the retinal blood vessels, although mention of this condition was made in many of the cases reported before and after operation, in only one case was it thought that there was any increase in size (de Takáts and Gifford's case 6).

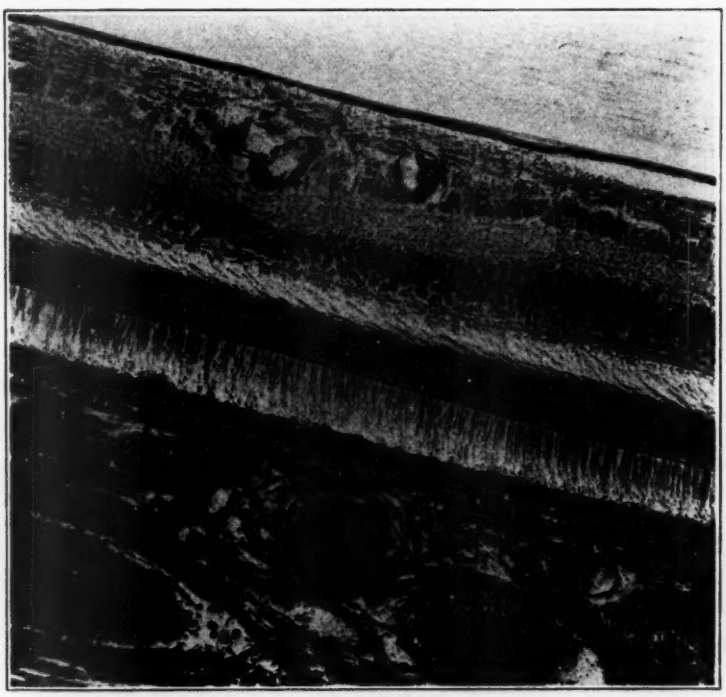

Fig. 2.—Photomicrograph of normal retina and choroid.

A brief reference to the pathologic basis of retinitis pigmentosa is of interest and of value in this connection. If the disease is due primarily to defects in the vascular system interfering with nutrition, it is conceivable that removal of vasoconstriction may be beneficial, particularly in the early stage of the condition.

If, however, degeneration of the vascular system of the eye is secondary to atrophy of the neuro-epithelium of the retina, it is difficult to imagine any benefit to be derived from sympathectomy. As the question stands at present, opinions are fairly equally divided.

Collins [23] placed retinitis pigmentosa under the heading abiotrophy of retinal neuroepithelium. He referred to the experiments of Wagenmann, who showed by division of one of the posterior ciliary arteries in the eyes of animals that the nutrition of the outer layers of the retina depends on the choroidal circulation. According to his studies, in the area supplied by the divided vessel both the choroid and the retina atrophied, the latter becoming pigmented and adherent to the former.

In the argument supporting his contention that retinitis pigmentosa originates in the neuroepithelium of the retina Collins cited a number of reported microscopic examinations of eyes with retinitis pigmentosa made by competent observers, in which the vessels of the choroid were

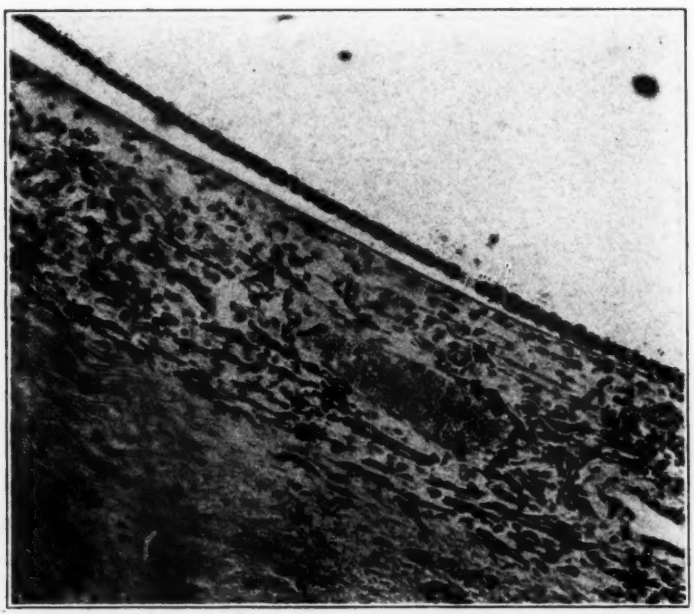

Fig. 3.—Photomicrograph of normal choroid, with normal pigment layer of the retina and normal sclera.

not changed from the normal, with the exception of a few defects in the choriocapillaris. In all these cases there was complete absence of rods and cones; the other retinal nervous elements were atrophic, and there was more or less proliferation of the neuroglia.

Collins stated:

> Atrophy of the percipient elements in a sensory organ is necessarliy followed by atrophy of the nervous elements connected with them, and a decrease in the vascular supply to the part. So with the retina in retinitis pigmentosa, following

23. Collins, E. Treacher: Internat. Cong. Ophth. **1**:103, 1922.

on the atrophy of the rods and cones, there is atrophy of the ganglion cells and other nervous elements in the retina together with a diminution of the smaller blood vessels of both the retina and choroidal circulation.

The pigmentation of the retina is due to the migration of pigment-epithelium cells into its substance; the reason why it is such a characteristic feature of the disease we term retinitis pigmentosa is that the disease commences in the rods and cones; the gaps left in the membrana limitans externa by their disappearance form tracks through which the pigment cells can make their way. We know that these pigment cells physiologically possess the power of ameboid movements. When stimulated by light they throw out filamentous processes between the outer segments of the rods and cones and on removal of the stimulus retract these processes into the body of the cell. They possess a capacity for positive phototactic movement which is kept in check only by their anatomic relations; when these

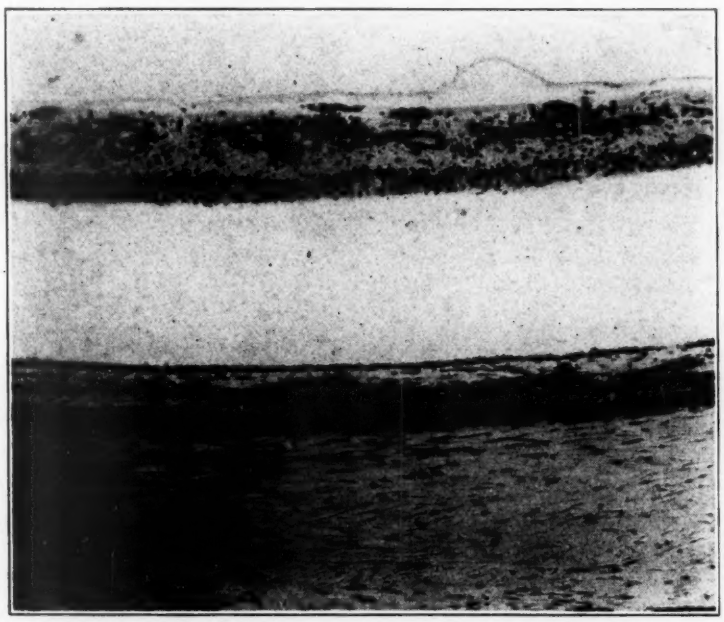

Fig. 4.—Photomicrograph of retina and choroid in retinitis pigmentosa.

are altered by the atrophy of the rods and cones together with the nerve elements of the retina, so that only a network of neurologlial tissue remains, then the pigment-epithelial cells attracted forward by the stimulus of light are able to make their way into the innermost layers of the retina where they accumulate in the lymph-spaces around the blood vessels and give rise to the characteristic branching patches.

In the cases of uncomplicated retinitis pigmentosa there is complete absence of evidence of inflammation; strictly spreaking, this condition is not retinitis.

A recent application of surgery to the sympathetic nervous system is section of the splanchnic nerves and lower dorsal sympathetic chain for the relief of systemic hypertension.

Fralick and Peet [24] reported:

The operation [as employed in the University Hospital] consists in resection of the greater and lesser splanchnic nerves and of the lower dorsal sympathetic chain, including the 10th, 11th and 12th ganglia. . . . The operation has always been performed bilaterally. Three objects may be attained by this procedure. First, and probably the most important, is the relief of renal vasoconstriction. . . . Relief of this vasoconstriction by splanchnic section has been followed not only by a permanent and often marked drop in blood pressure, but by marked improvement in kidney function.

The second object hoped for is the prevention of excessive adrenalin secretion by preventing stimulation of the suprarenal glands from higher centers.

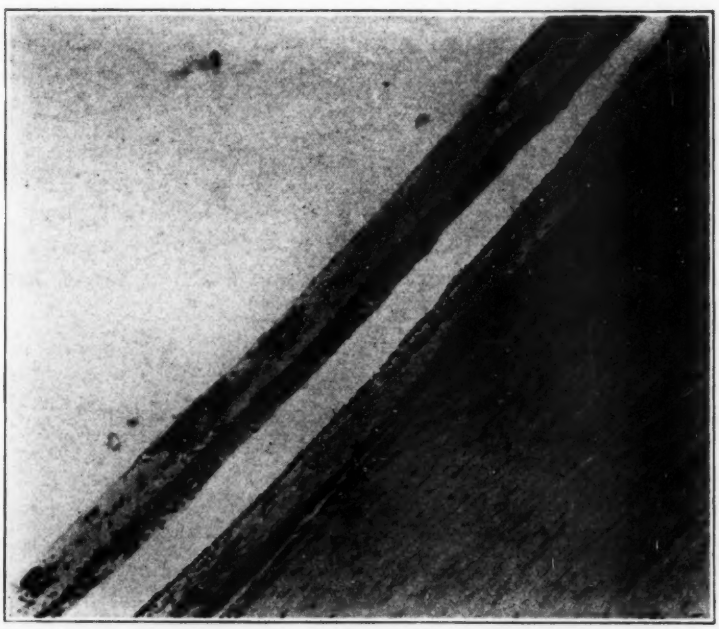

Fig. 5.—Photomicrograph of retina and choroid in retinitis pigmentosa.

The third object is the production of a great vascular bed incapable of vasoconstriction.

Operations on ninety patients are reported, in all of whom the preoperative systolic arterial tension was 200 or over. In forty-two of these the time that had elapsed since the operation, varying from five to twenty-two months, was sufficient to enable one to arrive at a reasonable estimation of the postoperative results; 14.4 per cent were apparently cured. Among those apparently cured were patients showing marked changes in the ocular fundus.

24. Fralick, F. B., and Peet, M. M.: Tr. Am. Acad. Ophth. **40**:119, 1935.

Fralick and Peet also reported:

> In the early stages of this operation it was expected that the fundus picture would offer us our best criterion as to operability. This idea was soon abandoned, since our prognosis as taken from the fundus findings was as often wrong as right.
>
> Those patients showing no appreciable fall in their blood pressure as a result of the splanchnic resection were not improved visually or objectively as far as the fundus findings were concerned except in one instance. . . . The 18 patients showing an appreciable drop in blood pressure were benefited ophthalmoscopically in only three instances; no change in the contracted retinal arteries was noted. In one patient there was no change in vision, but the edema of the disk and retina was markedly less, and the lipoids were nearly all absorbed fifteen months postoperatively. The other two cases presented marked improvement in vision but in only one of these was there any appreciable change in the fundus picture.

Other surgeons have reported improvement in a small percentage of cases of retinitis in association with systemic hypertension, after similar operations.

It has been abundantly demonstrated that changes in the size of the retinal vessels occur in normal eyes on resection of the cervical and first and second thoracic ganglions by the work of Wagener [25] at the Mayo Clinic in the treatment of Raynaud's disease by sympathectomy.

In regard to enophthalmos after sympathectomy, naturally, on the production of a pseudoptosis one acquires the impression that the eyeball behind partly closed lids is deeper in the orbit that its fellow. In normal conditions one eye may be located farther back in its orbit than its fellow, as measured with the exophthalmometer. Wagener,[26] in his excellent study of the subject, reported readings taken in ninety-four cases of Horner's syndrome; he found but one case of enophthalmos, and he opined that this condition may have been due to shrinkage of the orbital tissues.

In a high percentage of the cases in which the cervical and upper thoracic sympathetic ganglions are removed in the treatment of diseases of the upper extremities Horner's syndrome is produced. Dilatation of retinal arteries occurs as frequently as narrowing of the pupils and palpebral fissures, and dilatation of the retinal veins occurs somewhat less frequently. In most cases the dilatation of the retinal blood vessels is more transient than the narrowing of the pupils, although it has been seen to persist more than a year.

SUMMARY

From the evidence deduced from the literature on the subject I find that surgical intervention on the sympathetic nervous system has been proved to be of little value in the treatment of atrophy of the optic nerve.

25. Wagener, Henry P.: S. Clin. North America **11**:867 (Aug.) 1935.
26. Wagener, Henry P.: Am. J. Ophth. **17**:209 (March) 1934.

In cases of exophthalmic goiter operation on the sympathetic nervous system has been abandoned by the general surgeon for operations that afford better results.

In the treatment of glaucoma, which was thoroughly investigated during the decade following Jonnesco's first operations in 1898, sympathectomy has been abandoned.

The ocular vascular disturbances accompanying general systemic arterial hypertension would not, in themselves, warrant the extensive surgical procedure necessary to control systemic arterial hypertension. It is pleasing to know that improvement in the ocular vascular conditions sometimes follows sympathectomy performed for the purpose of reducing systemic arterial hypertension.

In the treatment of the disease known as retinitis pigmentosa it has been fully demonstrated that sympathectomy is of no value in advanced cases. The results of sympathectomy so far reported indicate that the operation *that cuts off all the fibers of the sympathetic nervous system that go to the eye* may retard or arrest the progress of the disease in the early stage.

In contemplating sympathectomy in the treatment of diseases of the eye it would appear to be desirable to ascertain the effect of temporary removal of the vasoconstrictor action of the sympathetic nervous system on the retinal blood vessels by the use of drugs or by testing systemic thermal changes. Careful measurements with the aid of the Morgan retinal graticule before and during the application of the tests referred to will decide the effect of operation on the blood vessels of the retina.

CORRECTION

In the article by Dr. Jacob B. Feldman entitled "Dark Adaptation as a Clinical Test," which appeared in the April issue (**17**: 648, 1937), the first part of the first sentence in the section "Sterility," on page 659, should have read: "Wilson and Dubois [17] observed at autopsy on a child with avitaminosis a change. . . ."

PHYSIOLOGY OF DISTURBANCES OF OCULAR MOTILITY

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. . . "In some ways we are so obsessed with the delight and advantage of discovery of new things that we have no proportionate regard for the problems of arrangement and absorption of the things discovered," Sir Josiah Stamp stated in his address as president of the British Association for the Advancement of Science in 1936.[1]

The facts and theories which are here presented are long established and should be better known and made a part of one's thinking about disturbances of ocular motility.[1a]

The anatomy of the parts concerned in ocular movements is well known. Attention is here called only to some anatomic facts which throw light on the widespread erroneous idea that in most cases heterophoria is due to *weakness* of one or more of the extra-ocular muscles.

The strength of a muscle depends on its cross-section. The amount of its shortening depends on its length. The ocular muscles measure from 8 to 18 sq. mm. in cross-section.[2] A square millimeter of striped muscle exerts a pull of 62 Gm.[3] when it contracts. Thus the ocular muscles are of such a size that, if all the fibers of a muscle contracted at once, the pull would be from 500 to over 1,000 Gm.

The weight of the eyeball is about 7 Gm. To this must be added the weight of the parts attached to it, which move with it: the conjunctiva, capsule, tendons, arteries, veins and nerves, especially the optic nerve. The eye does not move like the hip joint, as a smooth ball in a smooth socket. The surrounding tissues move with the eye,

1. Stamp, J.: The Impact of Science upon Society, Science **84**:235 (Sept. 11) 1936.

1a. There is no department of ophthalmology in which erroneous ideas are so prevalent as in that of ocular motility and binocular vision. As a result, elaborate superstructures are erected on flimsy foundations. These erroneous ideas are not limited to manufacturing opticians and optometrists, but the weeds have spread extensively among those who should know better. Instead of following the old advice and "letting the wheat and tares both grow together until the harvest," this effort is made to pull some of the weeds and cultivate and fertilize the grain.

2. Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1931, vol. 1, p. 203.

3. Howell, W. H.: Textbook of Physiology, ed. 13, Philadelphia, W. B. Saunders Company, 1936, p. 44.

but less and less as one gets farther from the eyeball. The tissues are packed in fat of the lowest melting point of any fat in the body (soft grease), which favors this motion. The total weight to be moved may be taken as from 12 to 13 Gm. The force required to move this weight at the speed with which the eye moves (10 degrees in three hundredths of a second) is less than 10 Gm.[4] Thus the muscles are strong enough so that only from one fiftieth to one hundredth of the fibers need to contract at one time to exert the necessary force. The way a muscle regulates the strength of its pull is by regulating the number of fibers which contract, since every fiber, if it contracts at all, contracts with its full maximum power—the "all-or-none" principle.[5]

Thus is provided an arrangement well calculated to take care of any demands for power likely to be made. It also makes provision for prolonged activity without fatigue, for only from one fiftieth to one hundredth of the fibers need to contract at one time, so that as the load is shifted from one set of fibers to another the first set have long since recovered from their fatigue before their turn comes again to take part in the contraction.

There is another important provision for effective mechanical work calculated to make the load easy for the muscles. Most of the muscles of the body are so attached, with respect to the fulcrum and the weight, that they are at a mechanical disadvantage from the point of view of power. Thus the biceps is attached about one twentieth of the distance from the elbow to the hand and so must exert a pull of a ton (1,000 Kg.) to sustain a weight of 100 pounds (45.4 Kg.) in the hand. The ocular muscles are attached to the surface of the eyeball at the maximum distance from the center of rotation (say, 12 mm.), and, owing to their attachment far in front of the equator, the pull is always tangent to the surface until the eye is rotated to the end of the contact arc. This contact arc is usually from 7 to 9 mm. long, which is equivalent to from 35 to 45 degrees. This is much more than the usual excursions of the eye demand. The almost incessant movements of the eye are for the most part within an excursion of 10 degrees from the primary position. Thus it is possible to reduce the contact arc in operations to, say, half its original length without seriously handicapping the ocular movements. If the contact arc is reduced to less than 3 mm. the result is rather crippling, and the reduction is warranted only when it is less objectionable than the condition it is aimed to relieve. If possible, it is better to keep a contact arc of 5 mm.

On the other hand, the mechanical advantage (leverage) is not increased when the contact arc is made longer by advancement. The

4. Lancaster, W. B.: South. M. J. **16**:38 (Jan.) 1923.

5. Lancaster, W. B.: "All-or-Nothing" Principle of Nerve Conduction and Muscle Contraction Applied to Eye, Tr. Sect. Ophth., A. M. A., 1923, p. 107.

muscle may be put on a stretch, but its mechanical advantage is the same as long as it pulls tangent to the surface of the eyeball. In tenotomy in which the attachment is moved farther back and the contact arc is shorter, the mechanical advantage rapidly diminishes *after* the eye rotates to the end of the contact arc and the pull of the muscle is no longer tangent to the eyeball.

Much stress has been laid by some writers on the importance of the limits of rotation of the eyeball (shown by the monocular field of fixation) as evidence of lack of strength of any ocular muscle. It is, of course, true that when a muscle is paralyzed rotation in that direction is greatly diminished, but in the case of paresis of slight or moderate amount it may be possible for the patient to rotate the eyeball in the direction of the paretic muscle to its normal extent. Striped voluntary muscles can contract to half their length. The ocular muscles, all of which are about 40 mm. long, are long enough to rotate the eye 20 mm. (90 degrees) from the primary position. Such an amplitude of rotation would play havoc with the vessels and nerves attached to the globe, and so check ligaments are provided to limit the rotation to about 40 degrees—half the rotation which muscles are capable of effecting. Since the muscle need exert only a small part of its total power to rotate the eyeball as far as these checks permit, it follows that a muscle may be definitely paretic and yet not show it by a limitation of *monocular* rotation in any direction. But, since the innervation to the two eyes is equal, the *binocular* rotations betray at once any paresis: (*a*) by the lagging of the paretic eye when the nonparetic eye fixes and still more (*b*) by the excessive rotation of the nonparetic eye when the paretic eye fixes (secondary deviation of the nonparetic eye).

The reason any provision is made for ocular movements is because all parts of the retina are not equally sensitive. The fovea so far surpasses the rest of the retina that if one wishes to look at anything one rotates the eye so that the image falls on the fovea. This is fixation and is the reason for the almost incessant movement of the eyes in the act of seeing.

A reflex in its most perfect form is entirely independent of the will and works best when consciousness has no part in it. Fixation is not a reflex of this sort. To be sure, the execution of the movements for the purpose of fixation is automatic, but the will has to decide what shall be the point of fixation. One decides voluntarily what to look at, and then the reflex is executed automatically. The neuromuscular mechanism for these movements is the heart of the subject discussed in this paper.

To take an example: A noise is heard which sounds like an airplane. One wills to look in the direction of the noise. The head (with the body)

is turned, and the image of the airplane falls on the retina. To be seen clearly the image must fall on the fovea; so the eyes (and head) are rapidly moved so that the eyes fix the airplane. A reflex requires an afferent impulse. In this case an auditory impulse occurred first, and the stimulus from a peripheral part of the retina occurred second. This retinal stimulus follows the visual pathway to the visual centers in the occipital lobes. Thence the path is to the psychomotor center nearby. Thence the efferent impulse goes to the supranuclear center for ocular movements, where coordination takes place. Here is decided the pattern of what muscles must contract and how much and what muscles must relax and how much. Obviously the pattern includes not only the extra-ocular muscles but the postural muscles of the neck and body. This is important in understanding the mechanism. It is easy to show that if any object attracts the attention and so fixation of it is willed, the necessary movement of the eyes is made by moving the eyes alone, if the distance to be rotated is less than 10 degrees.[6] If the distance is 15 degrees or more, the head is moved as well as the eyes. If the ophthalmologist tells a patient to look up, only the most sophisticated does so by rotating the eyes alone. Nearly all rotate the head more than the eyes.

Thus the supranuclear centers or groups of nerve cells and connections have an important function. From these cells go the messages to the nuclei of the various muscles, among them the ocular muscles, ordering certain muscles to contract and always, as an integral part of the action, ordering their antagonists to relax.

These supranuclear centers are capable of responding not only to messages from the psychomotor area of the visual center but also to messages from other sources. One of the most important of these centers is the voluntary center for ocular movements in the frontal lobe. Here the impulses arise from the will and are not purely reflex. Other impulses arise from the semicircular canals and from the proprioceptive centers. The labyrinthine control is designed to maintain, by compensatory movements of the eyes, a stationary field of vision, in spite of movements of the head. But the decision as to how a movement shall be executed—the pattern of the stimuli to be made use of—is automatically carried out by these supranuclear centers.

Yet the supranuclear activities play little or no part in the philosophy of the average ophthalmologist. He sees an exophoria and says that the lateral rectus muscles are pulling too hard or that the mesial rectus muscles are not pulling hard enough, which is true. But he jumps to the naïve conclusion that the former muscles are too strong or the latter too weak, whereas the muscles are probably faithfully carrying out the

6. Fischer, F. P.: Arch. f. Ophth. **113**:394, 1924; **115**:49, 1924.

orders brought to them by the nerves. The nerves, for their part, are merely transmitting the impulses from the nuclei, and the nuclei are issuing only such orders to contract or to relax as the messages from the supranuclear centers call for.

Any part of this neuromuscular mechanism may be out of order, and more than one part may be affected. But is it not reasonable to think that the most complicated part is the most likely to be acting imperfectly? When the patient is put through a course of orthoptic training, is it reasonable to suppose that the chief factor is a strengthening of the muscles already one hundred times as powerful as the task calls for? Does Paderewski play the piano so much better than another person because his muscles are stronger?

BINOCULAR MOVEMENTS

The two eyes move as the two parts of a single organ. Voluntary innervation of one eye alone is impossible under normal conditions, still less innervation of one muscle alone. It has been seen that to move one eye in any direction requires the cooperation of all six of its extraocular muscles and also of the postural muscles which move the head. It is even more important to grasp the fundamental unity of the binoculus. The innervation to the two eyes is always equally divided between them. In conjugate movements to the right, the right movers of both eyes are stimulated to contract and their antagonists to relax and the other muscles to steady the eyes as may be required. In disjunctive movements—convergence, divergence, sursumvergence, cyclovergence—both eyes are also equally innervated. For example, in convergence the two mesial rectus muscles contract, and the two lateral rectus muscles relax. Often combinations of conjugate and disjunctive movements are made at the same time, as when one looks at an object which is both nearer (convergence) and farther to the right (conjugate dextroversion) than the previous fixation point. And if it is also lower, conjugate downward rotation is added and, with this, normally a disjunctive conclination of both eyes (the vertical meridians rotating slightly inward at the top). It is possible by a simple trick to move one eye and hold the other practically still, but the innervation is still binocular, and the fact that one eye does not move is because it receives at the same time innervation, say, to converge and to rotate to the right (if it was the right eye that kept still), these two innervations being equal and opposite. The trick is to have the distant point of fixation and the near point of fixation both on a line with the right eye. Then the right eye does not have to move in shifting fixation from the distant to the near object.

If a prism base in is placed before the right eye while the two eyes are fixing a distant light, only the right eye moves, but both eyes

are innervated (divergence plus dextroversion). When the strongest prism which a patient can overcome has been placed before his right eye, it will be impossible to add another prism diopter before that eye without producing diplopia, and it will also be impossible to place a prism base in before the left eye without producing diplopia. To overcome any prism added to the left eye would require further divergence, but the prism before the right eye already exhausted the power of prism divergence of the binoculus. This shows the great fundamental difference between the capacity of the eyes to rotate outward in divergence (a binocular movement) and in abduction (a monocular movement) when the other eye is covered. It is better to speak always of convergence and of divergence when that is what one means, to speak of dextroversion and levoversion when one means conjugate movement of both eyes to the right or the left, and to reserve the terms adduction and abduction for movements of one eye (the other being covered or disregarded). The fundamental importance of this is apparent when it is found that either eye can be adducted or abducted 40 degrees or more but that the two eyes can be converged much less than that and diverged only from 3 to 4 arc degrees. The poor divergence is evidently not due to weakness of the muscles, since the same muscles can rotate the eyes 40 degrees when acting conjugately, though they can rotate the eyes only one-tenth as much when acting disjunctively.

The mechanism by which the two eyes are made to work together as a binoculus is the fusion faculty. It has been seen that there is a neuromuscular mechanism for fixation. Movements for fixation made by the eyeball and the head are executed with great precision and speed. Obviously the adjustment or coordination must be still more delicate and adaptable to varying conditions in the case of binocular movements in which the eyes must both at the same time fixate so that the images in the two eyes will fall on corresponding points of the retinas. The penalty for any lapse is diplopia. The antipathy to diplopia (diplopiaphobia)[7] may be regarded as the driving force behind fusion.

Of course, fusion includes much more than simply the simultaneous correct fixation of the two eyes on the object looked at. Just as there are two images on each retina, so there are two sensations of vision, one from each eye. When the images are on noncorresponding (disparate) areas there are two sensations and two perceptions, or diplopia (unless the disparateness is such in size and kind that fusion can take place). When the two images fall on corresponding points the two sensations give rise to a perception which (if the two sensations are

7. van der Hoeve: Operationslehre, in von Graefe, A.; Saemisch, T., and von Hess, C.: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1922, p. 1589.

alike and indistinguishable under introspection) does not differ from either of its components. But if the two sensations differ slightly, either because they fall on slightly disparate areas or for some other reason (for example, one may be brighter than the other or of a different color), the resulting synthesis of the two sensations is a percept which differs from each. It is not the algebraic sum of the two but is an entirely new perception. If the two cannot be synthesized (if fusion is impossible) one gets retinal rivalry: First one of the sensations assumes preponderance while the other is suppressed, and then the suppressed one engages the attention and the other is in abeyance.

In other words, there are a motor side and a sensory side to fusion. The latter is the larger and more fascinating part of the subject, but attention must not be diverted from the motor side, which is the concern of the present paper.

The path of the reflex for the correction of diplopia—the fusion reflex—is from the retinas to the visual cortex, where the sensation of diplopia reaches consciousness. Without consciousness the reflex does not act. From the visual centers the efferent impulses to correct the diplopia go to the psychomotor centers, the supranuclear centers, the motor nuclei, the peripheral nerves and the muscles.

Both eyes are equally innervated, and suitable combinations of conjugate and disjunctive movements result in bringing the images onto corresponding points so that fusion is possible.

The ability of the eyes to make the motor adjustments necessary for fusion is measured by the amplitude or range of fusion. The capacity of the eyes to diverge in the interest of single vision is the amplitude of divergence. It is measured in two ways: (1) by some form of haploscope, such as the amblyoscope or the synoptiscope, and (2) by overcoming prisms base in (prism divergence). Similar tests are made of sursumvergence and deorsumvergence. These movements are automatic and not under voluntary control. When the attention is directed to the object the necessary adjustment takes place. Convergence may be measured in the same ways but also in a third way by moving a fixation object nearer and nearer to the eyes until the near point of convergence is reached. When fixing in this way, the eyes are not only converging as the object is brought nearer, but also accommodating. Thus there are two kinds of convergence, one in which the eyes converge and at the same time accommodate, called accommodation convergence, and the other in which the convergence is not associated with accommodation but the focus is kept on a distant object. This is usually done by prisms placed before the eyes while they are fixing and focusing a distant object, and so this form of convergence is called prism convergence.

Since accommodation is so intimately and habitually associated with convergence, it is possible for the eyes to converge more easily and to a greater degree if, at the same time, accommodation is permitted and encouraged. Prism convergence therefore has a much smaller amplitude than accommodation convergence. It is possible with a little practice to exercise an accommodation effort while fixing but not focusing a distant point of light and overcoming prisms base out. If the patient tries to imagine that the point being fixated is close to his face and exerts his accommodation, disregarding the fact that the fixated point becomes out of focus when he does so, he can soon learn the trick of overcoming much stronger prisms base out. Thus his initial prism convergence may be only 15 prism diopters, while after a few days' practice it may be 40+ prism diopters. In the ordinary use of the eyes, if a person has exophoria for distance, he overcomes it by his fusion power (convergence), but obviously not by his accommodation convergence, since that would throw out of focus the objects at a distance at which he is looking. Hence prism convergence is more important in exophoria for distant vision. On the other hand, in exophoria for near vision, it is, of course, allowable, and even necessary, to accommodate, and so accommodation convergence is significant in exophoria for near vision (often called insufficiency of convergence).

In regard to the subject of heterophoria, much has been written about the position of the eyes at rest. This introduces a misleading conception. What one measures in measuring heterophoria is not the deviation when the eyes are at rest, because as a matter of fact, they never are at rest; that is, the muscles are never inactive, at rest, totally relaxed, not contracting. It is easy to show that even in sleep and in narcosis the ocular muscles are in a state of tonus, while after death they contract in rigor mortis. Even if one *could* determine the position of the eyes when all the motor nerves to all the ocular muscles are completely paralyzed, this would not clarify the problem of heterophoria. It is essential for a correct conception of heterophoria to keep in mind the fact that the ocular muscles are in a state of tonus, that is, they are being innervated. The position of the eyes, in short, is determined not simply by the anatomic conditions, but by these as modified by innervation of the muscles. What one does in measuring heterophoria is to determine the position of the eyes when fusion is eliminated, not when *all* muscular activity is eliminated. It is the fusion-free position, not the position of rest. Hence it is important to consider tonus.[8]

All the striped muscles are normally in a state of tonus. It is a reflex affair complicated and not as well understood as could be wished. In the case of the ocular muscles, besides the sources of tonus shared

8. Cords, in Schieck and Brückner,[2] vol. 3, p. 470.

by other voluntary muscles, such as the vestibular (which is static from the otoliths and dynamic from the semicircular canals), the proprioceptive and the central, there is the special source from the retina. Stimulation of the retina causes increase of ocular tonus. But there is another, even more important aspect of the fusion-free position. When one eye is covered and the other eye fixates, stimuli go to the muscles of both eyes. If the fixing eye accommodates for any reason (e. g., to correct hypermetropia), convergence is stimulated. It is not to be expected that these stimuli can be so distributed to the two eyes that perfect parallelism (orthophoria) would always result.

The sum of the matter is that nerve stimuli are constantly flowing to the extra-ocular muscles, and the stream varies with varying conditions. The resultant tonus or contraction of the twelve muscles does not produce a perfect balance, and the fusion faculty has to correct any shortcomings. When fusion is eliminated the ophthalmologist is able to see how much work the fusion faculty was doing before he covered one eye or otherwise eliminated fusion. This is the important thing about heterophoria—it shows what the task of the fusion faculty is. The measurement of the amplitude of fusion shows the capacity of the fusion faculty to deal with this. Treatment is concerned with (*a*) reducing the task of the fusion faculty (reducing the heterophoria by operation, by prisms, by plus or minus lenses, or evading it by wearing a patch over one eye), or (*b*) increasing the ability of the fusion faculty (by improving the general health, by orthoptic exercises or by operation).

Another important fact about tonus is the tendency for it to persist an appreciable, and often a considerable, time after the stimulus is removed. For example, in measuring the amplitude of fusion prisms base down are placed before the right eye, and increased up to the maximum that can be overcome, and time is allowed for the adjustment to be made; a 3 diopter prism placed abruptly before the eye may cause diplopia, but if, by a rotary prism or otherwise, the strength is slowly and gradually increased from 0 to 3 diopters in the vertical direction, it can perhaps be readily overcome. Another way is to start with the prism base out (or in) and slowly rotate it until it is held base down. This gradually increases the vertical power from 0 to 3. If the prism is then reversed and the amplitude measured base up, the amount that can be overcome, even if the strength is gradually increased, will be less than it would have been if the amplitude base up had been tested first. After repeating the test with the prism base up a few times or after waiting long enough, the true value can be obtained. The reason is that the effort to overcome the first prism, the prism base down, has produced a tonic contraction (it might be called a spasm), which persists for an appreciable, and often a considerable, time. Much

depends on how long the effort is maintained to overcome the first prism. If it were worn for twenty-four hours, definite hyperphoria persisting for some time would be found. Now let it be supposed that a patient has a condition the correction of which requires a considerable part of his amplitude of fusion. This may have existed for years, producing a strong correcting tonus. He is given prisms which correct a part of this. Then after a short period of time has elapsed it is found that he needs stronger prisms. These are given, and again he needs stronger prisms. This common experience has been interpreted by many ophthalmologists as showing the folly of giving prisms at all, since, they have said, this only increases the defect. The true explanation is that the first prisms corrected only the manifest part of the deviation, and when the strain on the adjusting mechanism was reduced some of the latent deviation, due to the tonus or spasm of the muscles making the adjustment, became manifest as the necessity for the spasm was removed. The same thing occurs in hypermetropia. The correction of the manifest portion of hypermetropia results in the appearance of what before was latent. So, far from being an argument against giving plus lenses for hypermetropia, this is the strongest evidence of the need of them.

Fusion at birth is imperfect. The process by which it is acquired is, like that of other habits, a facilitation of reflex paths (compare the conditioned reflexes of Pavlov). It is built up on an already existent basis by education and practice, but not to the same extent in all persons. It is this capacity for education and development of the function that is made use of in orthoptic training. Any faculty which is present in some degree can be amplified and perfected. If it is not present it cannot be conjured into existence by apparatus.

When the established relations of the two eyes to each other are changed by the experiment of placing a prism (not too strong) before one eye, not only is the tendency to diplopia promptly corrected, but all movements are immediately executed correctly, although this, of course, requires an entirely new set of coordinations—an entirely new set of neuromuscular impulses to move the eyes from one fixation point to another. Similarly, if an exostosis grows in the orbit, displacing the eyeball, the neuromuscular mechanism modifies its impulses to the different muscles and maintains binocular single vision, even when the displacement amounts to several millimeters. This shows clearly that the neuromuscular mechanism for binocular movements is not a fixed and rigid mechanism, but one that is of marvelous adaptability to a fairly wide range of conditions. It is not as though there were in the brain a certain button to be pressed (a center to be stimulated), causing the eyes to move up to the right, another button to be pressed, caus-

ing movement down to the left, etc. A slight change in the position of the head or the wearing of lenses, especially prisms, would throw this rigid mechanism entirely out of gear. The actual mechanism is an adaptable machine which automatically and instantaneously makes allowances for all such complications and executes the movement with almost incredible speed and accuracy in spite of them. This capacity for compensation for any interference with the regular functioning of the mechanism shows how inadequate is the explanation offered by some authors for heterophoria and its consequences, viz., slight anatomic variations in the attachments of the muscles or the structural details of the orbit. Such handicaps are taken care of by this mechanism promptly and efficiently. One must look for trouble not in such simple factors but in some derangement of the "neuro" part of the neuromuscular mechanism, especially in the supranuclear centers for coordination. The purpose in operating[9] on the ocular muscles in heterophoria is not simply to correct some possible (hypothetic) anatomic or mechanical factor causing imbalance, but rather to change the position of the eyeball in the orbit so that there is less adjustment necessary on the part of the neuromuscular mechanism—the fusion faculty. For instance, if there is exophoria of 6 diopters for distant vision and 16 diopters for near vision, the neuromuscular mechanism can correct it (compensate it). If not, the condition would be not a phoria but a tropia. The task of executing the incessant movements of the eyes, while the neuromuscular mechanism is handicapped by the necessity of correcting the phoria, is greatly diminished if the position of the eyeballs is so changed that the adjustments start from zero or near zero, i. e., if there is no phoria to be provided for by the neuromuscular mechanism while the binocular movements are executed. The notion that the operation is designed to strengthen a too weak muscle or weaken a too strong one is very wide of the mark and based on ignorance of the physiology of ocular movements.

9. Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 1133.

LESIONS OF THE FUNDUS

IN ESSENTIAL HYPERTENSION AND IN ARTERIAL AND RENAL DISEASES

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Recent studies of arterial diseases have shown the importance of familiarity with the various vascular lesions in the fundus, their microscopic appearance and their interpretation with respect to the clinicopathologic diagnosis. It is necessary, therefore, that the ophthalmologist possess as a working basis a clinicopathologic classification which is acceptable to both the clinician and the pathologist.

Important contributions on the ophthalmologic phase of vascular diseases have been presented in the past few years by Wagener,[1] Benedict,[2] H. Friedenwald and J. S. Friedenwald,[3] Evans[4] and others. Numerous researches, carried out from a general pathologic point of view, are still being made. Unfortunately, clinicians and pathologists are not entirely in agreement on some salient points.

Valuable aid in the differential diagnosis and prognosis of arterial diseases is afforded the clinician by the presence or absence of lesions of the fundus. Vascular lesions and their effects on surrounding tissues can be observed solely in the eyegrounds; this emphasizes the importance of examinations of the fundus. Essentials in these examinations are: full dilatation of the pupils, maximum illumination from the ophthalmoscope, proper lenticular correction in the ophthalmoscope and a knowledge of physiologic variations, especially of the vasculature. The red-free light of Vogt is of great assistance, and I have imitated this

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From the Department of Ophthalmology of the New York Post-Graduate Medical School and Hospital (Columbia University).

1. Wagener, H. P.: The Retinitis of Malignant Hypertension, Tr. Am. Ophth. Soc. **25**:349, 1927.

2. Benedict, W. L.: Retinitis of Cardiovascular and of Renal Disease, Am. J. Ophth. **4**:495, 1921.

3. Friedenwald, H.: Pathological Changes in the Retinal Blood-Vessels in Arteriosclerosis and Hypertension, Tr. Ophth. Soc. U. Kingdom **50**:452, 1930. Friedenwald, H., and Friedenwald, J. S.: The Retinal Blood Vessels in Hypertension and Arteriosclerosis, Bull. Johns Hopkins Hosp. **45**:232, 1929.

4. Evans, J. N.: An Interpretation of Defects in the Visual Field, Arch. Ophth. **3**:153 (Feb.) 1930.

procedure by using a green filter in the revolving disk of the ophthalmoscope. With this method blood vessels, hemorrhages and white foci stand out in marked contrast to the surrounding area. J. S. Friedenwald uses a giant ophthalmoscope, and Evans employs angioscotometry for a more minute study of the blood vessels. Slit lamp studies of the blood vessels of the iris in animals and of the sclera and conjunctiva in man have not as yet proved of practical value. Each fundus should be carefully examined, as an isolated hemorrhage in either may exist as an index of arterial disease. Vascular lesions are not inflammatory; therefore, such terms as retinosis and retinopathy, rather than retinitis, are now being used to describe them.

In the records of cases of vascular diseases of the New York Post-Graduate Medical School and Hospital the following facts are noted:

1. The presence or absence of ocular lesions.

2. A description of the lesions of the fundus.

3. A diagnosis of the condition of the fundus, to which is added the diagnosis of the internist.

4. Comment with respect to the lesions of the fundus and their relation to the clinical facts.

The diagnosis of the condition of the fundus in these cases is so variously recorded that my associates and I have adopted, for the present, a descriptive classification based on direct observation, using the following terms:

(*a*) Papillo-arterial sclerosis or spasm (moderate or severe) in: essential hypertension (benign or malignant), nephritis, etc.

(*b*) Papilloretinal arterial sclerosis or spasm (moderate or severe) in: (the disease being stated, as in *a*).

(*c*) Retinal arterial sclerosis or spasm (moderate or severe) in: (the disease being stated, as in *a*).

(*d*) Choked disk, with or without retinal arterial sclerosis or spasm in: (the disease being stated, as in *a*).

The following diseases will be briefly considered: (I) essential hypertension (benign or malignant); (II) generalized arteriosclerosis; (III) diabetes; (IV) nephritis.

I. ESSENTIAL HYPERTENSION

By many authorities essential hypertension is considered a clinical entity, because other diseases are not present to an extent sufficient to account for the markedly increased and persistently high blood pressure. General arteriolar sclerosis or spasm is usually present. The organs

involved are the eyes, kidneys, brain, heart and others. Angiospasm of the small arteries of the eye and other organs, particularly the kidneys, is by some considered to be the initial factor in the narrowing of the lumen of these vessels, which may result eventually in sclerosis. The cause of this spasm is regarded by some as a vasomotor disturbance due to hyperfunction of the adrenal glands, while others maintain that it is due to a toxic factor such as is observed in the toxemias of pregnancy. In the latter condition localized contraction of retinal arteries is not fixed, but, according to Mylius,[5] can be seen to travel in a peristaltic fashion. After delivery the hypertension is gradually relieved.

Arterial contraction results in venous stasis, causing various grades of pathologic changes of the fundus in all types of lesions of the fundus. To illustrate this condition I shall present two drawings from photographs of the fundus of a 12 year old boy with unilateral ethmoiditis (case 21), one taken before surgical intervention, the other taken three weeks later. One fundus showed a marked choked disk with a star figure on one macula, retinal venous stasis and pronounced arterial contraction, hemorrhages and infiltrates, but no general hypertension. Vision equaled counting of fingers at 6 inches (15 cm.). The ophthalmoscopic changes were unilateral, resembling the findings, in cases of renal disease or tumor of the brain occurring without a macular lesion. Following operation on the ethmoid and the sphenoid sinus there were, within three weeks, a normal fundic picture and normal vision. The facts of this case seem to confirm also the theory that a local toxic factor may cause arterial spasm and venous stasis. Of course, further similar observations are essential for definite confirmation.

Essential hypertension exists in a benign and a malignant form, and there are also borderline conditions. Whether the benign form usually develops into the malignant form is still under investigation.

In the benign form the moderate hypertension may exist for a great many years, and lesions of the fundus are usually absent or, if present, are mild and progress slowly. With proper care the prognosis is favorable.

Malignant essential hypertension with marked and long-standing increase of the blood pressure occurs in both adults and children. In the latter it is rare and is usually fatal within a few months. In addition to high blood pressure, the principal symptoms are headaches and vomiting, disturbance of the renal function not appearing until the approach of the terminal stage. The ophthalmoscopic findings are similar to those in cases of cerebral tumors occurring without macular lesions, or in cases

5. Mylius, K.: Funktionelle Veränderungen am Gefäss-system der Netzhaut, in Abhandlungen aus der Augenheilkunde und ihren Grenzgebieten, Berlin, S. Karger, 1928, no. 10. (Illustrated with photographs of fundi.)

of chronic glomerular nephritis, although there is no clinical evidence of these diseases. Lesions of the fundus were present in a case of malignant hypertension in a 10 year old girl seen at the New York Post-Graduate Medical School and Hospital, who died of uremia due to mild nephritis.

Adults with malignant hypertension usually show marked varying lesions of the fundus.

The lesions of the fundus act as a prognostic indicator in malignant hypertension. Some patients who show marked retinal lesions live from three to five years after discovery of the lesions. If edema of the disk occurs it indicates renal complications.

II.- GENERAL ARTERIOSCLEROSIS

The lesions of the fundus in generalized arteriosclerosis or arteriolar sclerosis are due to sclerosis of varying degrees which affects the retinal and choroidal vessels, causing hyaline and fatty degeneration of their intima and media. These changes affect parts of the walls of the vessels, causing encroachment on the lumen and resulting in endarteritis. In this condition hypertension is a secondary factor; in fact, it may be absent if the fundic findings are mild, though it is usually present if the fundic findings are severe. These fundic findings are extremely important to the patient and the clinician. Apoplexy is due to sclerosis, thrombosis and embolism affecting the cerebral vessels. These conditions are frequently associated with coexisting retinal arteriosclerosis and other ocular manifestations. Arteriosclerotic lesions are exceedingly diverse in their distribution, one vessel (or a part of it) or certain regions being affected, while others escape. In fact, the retinal vessels in cerebral sclerosis are frequently normal. Disease of the retinal vessels of local origin is not a sure sign of disease of the cerebral vessels; it simply furnishes a presumption in favor of its existence, particularly since the eye is a part of the brain. Disease of the retinal and choroidal vessels may exist without any ophthalmoscopic signs, as has been proved by microscopic examination; nevertheless, the retinal vessels provide the best guide one possesses as to the general state of the vessels.

In retinal arteriosclerosis the progress of the ocular condition is of secondary importance compared to the condition of the kidneys, brain and heart, for the prognosis as regards life depends on the condition of those organs. Death usually results from apoplexy within four years after the recognition of marked retinal arteriosclerosis.

In the senile type of arteriosclerosis, seen in older people, the fundus frequently shows simply varying degrees of arteriosclerosis. A small percentage of patients show hemorrhages, whitish patches and glistening white spots and, more rarely, edema of the disk.

III. DIABETES

Diabetes, being a disease of middle life, occurs at a time when arteriosclerosis is likely to be present. The ocular complications usually appear only after the disease has progressed for some years and when the patient is therefore debilitated and consequently more susceptible to chronic disorders. The bilateral fundic findings are so well known that they require no description, except that the retinal vessels show definite evidence of varying degrees of arteriosclerosis. Hypertension, if present secondarily, is likely to aggravate the lesion of the fundus. With others who have studied so-called diabetic retinitis, I believe that this lesion is due mainly to sclerosis of the retinal and choroidal blood vessels and that the disturbance of the sugar metabolism, as I have previously reported, is contributory only under certain conditions:

1. Thickening of the vascular walls is demonstrable microscopically in some cases although the walls appear normal when examined ophthalmoscopically.

2. Hemorrhages may be due to degeneration of the vascular walls, not to hypertension. In many of the cases which were observed for long periods, there was no hypertension, venous stasis or cellular disturbance to explain the presence of extravasated blood in the retinal tissues.

3. In a certain number of cases, although the general condition improves under dietetic treatment and treatment with insulin, the retinal pathologic process increases, indicating the presence of a permanent pathologic vascular condition.

4. In lipaemia retinalis occurring during severe diabetes in the young, a marked alteration of the sugar metabolism is present in the blood, and all case reports which include a record of microscopic examination mention the observation of normal blood vessels.

5. Hypertension and arteriosclerosis are frequently associated with diabetes.

6. Diabetic gangrene is due primarily to arteriosclerosis.

7. Death of a person with diabetes is often caused by apoplexy, owing to cerebral arteriosclerosis.

8. The retinal arteriosclerosis is part of the general arteriosclerosis.

These facts lead me to believe that so-called diabetic retinitis is due mainly to arteriosclerosis of the retinal and choroidal blood vessels and that in some instances diabetes may influence the picture, particularly in the cases in which the disease is protracted. Diabetic retinitis may, therefore, be called retinal arteriosclerosis (moderate or severe) in association with diabetes, rather than diabetic retinitis. The optic disk is rarely involved in uncomplicated diabetes.

IV. NEPHRITIS

Renal diseases are variously classified as: acute nephritis, chronic or diffuse glomerulonephritis, benign or malignant nephrosclerosis (Volhard [6] and Fahr [7]) and renal arteriosclerosis or arteriosclerotic nephritis. The term chronic interstitial nephritis is now generally regarded as lacking a pathologic basis; the terms usually considered are acute and chronic (diffuse) glomerulonephritis and renal arterial sclerosis, the basic lesion in the kidney being an arteriolar lesion of the renal vessels. In generalized arteriosclerosis the renal vessels take part in the distribution of the vascular lesion, although for some time there may be no marked involvement of the tubules or glomeruli, and the fundic picture shows retinal arteriosclerosis. The fundus is not involved in acute nephritis or nephrosis following scarlet fever, mercury bichloride poisoning and some types of toxemia of pregnancy.

In chronic glomerulonephritis the glomeruli are markedly involved, and the arterioles show sclerosis, with the production of a generalized toxic factor which Volhard considers responsible for the lesions of the fundus. When malignant essential hypertension and arteriolar sclerosis accompany chronic glomerulonephritis, or vice versa, it is impossible, as a rule, to state whether the fundic picture represents malignant essential hypertension or glomerulonephritis or both. The clinical history will frequently decide the diagnosis, but not often the sequence, of the disease.

The prognosis of acute nephritis is usually favorable; that of chronic glomerulonephritis is grave, death being usually due to uremia. The disease may continue for two or three years or longer after the discovery of the lesions of the fundus. The condition of the fundus is generally known as albuminuric neuroretinitis, but a more tenable term is papilloretinal arteriosclerosis (severe) in association with chronic glomerulonephritis, since a number of authorities deny the presence of inflammation as determined by microscopic studies of the fundus and also because, for obvious reasons, albumin is not considered to be an etiologic factor.

In a previous report by me [8] of a study made in cooperation with Dr. I. J. Levy and Dr. Larkin, of eighteen cases of nephritis, including arterial disease, in which the eyes and kidneys were simultaneously studied, I stated that the basic cause of the changes of the fundus was

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6. Volhard, F.: Netzhautveränderungen und Nierenleiden, Zentralbl. f. d. ges. Ophth. **5**:470, 1921; Die Pathogenese der Retinitis albuminurica, ibid. **21**:129, 1929.

7. Fahr, T.: Ueber Nephrosklerose, Virchows Arch. f. path. Anat. **226**:119, 1919.

8. Cohen, M.: Significance of Pathologic Changes in the Fundus in General Arterial and Kidney Diseases, Tr. Sect. Ophth., A. M. A., 1922, p. 60

primary chorioretinal arteriosclerosis. The edema of the disk, the hemorrhages, the whitish foci and other changes resulted from this cause, either alone or in association with some unknown factor. The degree of arteriosclerotic change disclosed by microscopic examination was not in direct proportion to the amount of pathologic change noted clinically in the fundus. In some cases the ophthalmoscope revealed no signs of retinal arteriosclerosis, though microscopically such a condition was demonstrable. The choroidal arteriosclerosis was more marked than the retinal and was sometimes present even when the latter was absent. Arteriosclerosis, together with definite edema of the disk, hemorrhages and whitish foci, in nephritis suggested that the kidney had already undergone contraction and was structureless. In the absence of pronounced edema of the disk, the kidney was usually the site of vascular changes alone.

At times it is difficult or impossible for the ophthalmologist to diagnose renal or other arterial diseases without the cooperation of the internist. On the other hand, an accurate knowledge of the lesions of the fundus is a valuable guide in the diagnosis, prognosis and care of these diseases.

REPORT OF CASES

I shall now present a brief clinical report of cases from various hospitals, including the average findings for the blood pressure and the blood chemistry. These case reports were accompanied by drawings of the fundus, as well as drawings from photomicrographs illustrating vascular diseases, showing sections of the eye and kidney. The drawings were of one eye only in each case, the observations in the other eye being similar.

CASE 1 (New York Post-Graduate Medical School and Hospital).—V. C., a girl aged 10 years, was admitted with the general diagnosis of essential hypertension (the malignant phase). There were a history of headaches and persistent high blood pressure for the past three years and a systolic murmur. The urine contained a slight amount of albumin and a few red blood cells. The urea nitrogen content of the blood was 11.2 mg. per hundred cubic centimeters. Examination of the spinal fluid revealed markedly increased pressure. The blood pressure was 190 systolic and 150 diastolic. The Mosenthal test showed normal fixation of specific gravity.

Edema with nephritis finally developed, and the child died of uremia four months after the drawing of the fundus was made.

Fundic Findings.—These consisted of bilateral papilledema, engorgement of the retinal veins, contraction of the arteries and a star figure in the macular area.

CASE 2 (Harlem Hospital).—C. H., a Negro woman aged 35, was admitted with the general diagnosis of essential hypertension (the benign phase). There was a history of glycosuria, hemiplegia on the right and rigidity of the neck for the past two months. The blood pressure was 180 systolic and 94 diastolic. Urinalysis showed that the urea nitrogen content was 9.4 mg. per hundred

cubic centimeters; the creatinine content, 1.3 mg., and the sugar content, 101 mg., and that albumin (3 plus) was present.

Fundic Findings.—There was a large preretinal hemorrhage.

CASE 3 (Sydenham Hospital).—S. G., a Negro woman aged 38, was admitted with the general diagnosis of essential hypertension (the malignant phase). The blood pressure was 250 systolic and 160 diastolic, and the urea nitrogen content of the blood was 21.7 mg. per hundred cubic centimeters. On account of the protracted high blood pressure and severe headaches, a partial unilateral adrenalectomy was performed, which temporarily relieved the symptoms. The patient was readmitted for a similar operation on the other side, but owing to the development of hemiplegia this was not performed, and after several weeks she was discharged.

At the time of the operation a small section of abdominal muscle and kidney tissue were removed for microscopic examination. This revealed a slight hyalinization of the glomerular vessels and a cloudy swelling of the tubular epithelium. The smaller renal vessels were strikingly hyperplastic. The vessels in the section of the muscle showed hyperplastic medial thickening.

Fundic Findings.—The veins were congested and the arteries contracted. There were fatty and white spots, and the macular lesion showed a partially formed star figure.

CASE 4 (New York Post-Graduate Medical School and Hospital).—T. R., a man aged 32, was admitted with the general diagnosis of essential hypertension (the malignant phase). With the exception of headaches for three months, there was no history of illness, and there was no evidence of dyspnea, precordial pain, cough, edema or hematuria. The blood pressure was 266 systolic and 170 diastolic. The urea nitrogen content of the blood was 15.5 mg. per hundred cubic centimeters, and the cholesterol content, 245 mg., and albumin (3 plus) was present in the urine. The Mosenthal test revealed nothing abnormal, and the Wassermann reaction was negative. There was no renal lesion. The patient was discharged after three weeks in the hospital.

Fundic Findings.—These consisted in edema of the disk, engorgement of the veins, hemorrhages, white spots and macular lesions.

CASE 5 (Bellevue Hospital).—E. B., a man aged 32, was admitted with the general diagnosis of essential hypertension (the end-stage of the malignant phase). He had been well until two and a half weeks previously, at which time headaches, vertigo and polydipsia developed. His heart was enlarged. His blood pressure was 190 systolic and 105 diastolic. The nonprotein nitrogen content of the blood was 250 mg. per hundred cubic centimeters; the creatinine content, 8 mg., and the hemoglobin content, 70 per cent. At the time of his admission the renal function was normal, and it remained so for over a month, when subacute glomerular nephritis developed, the primary condition being malignant essential hypertension. The patient died after two and a half months in the hospital.

Fundic Findings.—There was general engorgement of the veins, with thrombosis of the central vein. Other findings were contraction of the arteries, hemorrhages, and edema of the disk and the surrounding area.

CASE 6.—C. C., a woman aged 52, was admitted with the general diagnosis of essential hypertension (the malignant phase). There was a history of hypertension for a year and a half, a condition which rest and rigid diet had failed to improve. The blood pressure was 210 systolic and 115 diastolic. The nonprotein nitrogen content of the blood was 89 mg. per hundred cubic centimeters; the urea nitrogen content, 11.5 mg.; the cholesterol content, 245 mg., and the sugar

content, 80 mg. There was no trace of albumin in the urine, and the Wassermann reaction of the blood was negative.

Fundic Findings.—There were a local profuse hemorrhage, with thrombosis, and whitish and white spots.

CASE 7 (Sydenham Hospital).—A. M., a Negro woman aged 45, was admitted with the general diagnosis of hypertensive cardiovascular disease. There was a history of rheumatic polyarthritis and attacks of shortness of breath during the previous year. Her heart was enlarged. The blood pressure was 280 systolic and 180 diastolic. The urea nitrogen content of the blood was 24.5 mg. per hundred cubic centimeters, and the hemoglobin content was 86 per cent. The Van Slyke urea clearance test showed a clearance of from 43.6 to 81 per cent of normal.

Fundic Findings.—The veins were engorged and tortuous, and the arteries were contracted. There was arteriovenous compression, and hemorrhages and white areas could be demonstrated.

CASE 8 (Harlem Hospital).—L. B., a Negro woman aged 28, was admitted with the diagnosis of hypertensive toxemia of pregnancy. The blood pressure was 180 systolic and 105 diastolic. The renal function was normal.

Fundic Findings.—With the exception of a small isolated elliptic hemorrhage covering a vessel, the fundus was normal.

CASE 9 (Sydenham Hospital).—F. G., a woman aged 24, was admitted with the general diagnosis of acute exacerbation of chronic nephritis due to toxemia of pregnancy. Her blood pressure was 240 systolic and 140 diastolic. The urea nitrogen content of the blood was 69 mg. per hundred cubic centimeters, and the creatinine content was 6.7 mg. Many red blood cells were present in the urine. The heart was normal.

Fundic Findings.—The arteries were apparently normal, but there were edema of the disk, congestion of the veins and whitish and white spots.

CASE 10 (Harlem Hospital).—M. T., a Negro woman aged 59, was admitted with the general diagnosis of arteriosclerosis associated with hypertension. Diabetes was also diagnosed, although the dextrose tolerance was normal. The blood pressure was 244 systolic and 130 diastolic. There was a trace of albumin in the urine, but microscopic examination gave negative results. The urea and creatinine contents of the blood were normal. The Mosenthal test showed some fixation of specific gravity.

Fundic Findings.—A light streak, resembling silver wire, was visible in the arteries. White spots could be seen in the macular area, and arteriovenous compression was present.

CASE 11 (Harlem Hospital).—L. L., a Negro woman aged 71, was admitted with the general diagnosis of arteriosclerosis associated with hypertension and nephritis, and myocardial damage. Her blood pressure was 200 systolic and 110 diastolic. The urea nitrogen content of the blood was 62.4 mg. per hundred cubic centimeters; the creatinine content, 2.6 mg., and the sugar content, 92 mg. Albumin (4 plus) was present. The hemoglobin content was 55 per cent.

Fundic Findings.—The principal findings were engorgement of the veins, arterial contraction and venous compression. Discrete white spots could be observed around the macular area.

CASE 12 (New York Post-Graduate Medical School and Hospital).—B. K., a woman aged 55, was admitted with the general diagnosis of diabetes and arteriosclerosis. The history showed that twelve years earlier the patient had complained

of polyuria and polydipsia. Examination at that time had revealed the presence of sugar in the urine. The blood pressure, on admission, was 158 systolic and 90 diastolic. The specific gravity of the urine was normal, and no albumin was found, but sugar was present (from 1.1 to 2.6 per cent).

Fundic Findings.—There were light streaks in the lumen of the arteries and discrete white spots in the macular area.

CASE 13 (New York Post-Graduate Medical School and Hospital).—G. K., a woman aged 58, was admitted with the general diagnosis of hypertensive retinitis accompanied by low grade chronic diffuse nephritis. She had a history of diabetes of twenty years' duration and poor vision for the last two years. The blood pressure was 215 systolic and 98 diastolic. The urea nitrogen content of the blood was 19 mg. per hundred cubic centimeters, and the nonprotein nitrogen content, 34 mg. Albumin (2 plus) was present in the urine. No sugar was found while the patient was under treatment.

Fundic Findings.—The disk was edematous. The arteries were contracted, some being entirely obliterated. The veins were tortuous and were bordered by a white line. Whitish spots, pigment and hemorrhages, both fresh and old, were also observed.

CASE 14.—A. N., a man aged 68 years, was referred to me with the general diagnosis of arteriosclerosis, hypertension and arteriosclerotic heart. His blood pressure was 150 systolic and 80 diastolic. The urine contained no sugar, but albumin (2 plus) was present. The creatinine content of the blood was 1.2 mg. per hundred cubic centimeters; the urea nitrogen content, 14.3 mg., and the uric acid content, 4 mg.

Fundic Findings.—The veins were engorged and tortuous, and there was thrombosis of the inferior temporal vein. The arteries were contracted. There were sclerosis of the choroidal vessels and arteriovenous compression. Macular lesions were also demonstrable.

CASE 15 (New York Post-Graduate Medical School and Hospital).—A. B., a woman aged 52, was admitted in a semicomatose condition, with a tentative diagnosis of tumor of the brain associated with homonymous hemianopia. After bitemporal decompression there was marked improvement in her general condition. Her blood pressure was 120 systolic and 70 diastolic and her renal condition normal.

Fundic Findings.—The fundi were normal, with the exception of an isolated arteriovenous compression which caused, at crossing, a change in the course of the vein. The findings were indicative of arteriosclerosis, probably accompanied by cerebral sclerosis.

CASE 16 (Manhattan Eye, Ear and Throat Hospital).—E. W., a woman aged 74, was admitted with the general diagnosis of senile arteriosclerosis and myocarditis. Her blood pressure was 180 systolic and 110 diastolic, and her renal function was normal.

Fundic Findings.—The veins were slightly engorged. The arteries were contracted and over certain areas were invisible, possibly because of their extreme thinness. The choroidal vessels in certain areas were sclerosed. There were signs of macular degeneration.

CASE 17.—A. G., a man aged 71, was referred to me with the general diagnosis of senile arteriosclerosis, myocarditis and Paget's disease. His blood pressure was 170 systolic and 90 diastolic. Examination of the blood and urine showed nothing abnormal. This patient died one month later, of myocardial failure.

Fundic Findings.—The veins and arteries were contracted, but only slightly. The choroidal vessels showed sclerosis over a large area. Lesions were visible in the macula, which also showed evidences of a previous large hemorrhage.

Case 18 (New York Post-Graduate Medical School and Hospital).—N. L., a man aged 37, was admitted with the general diagnosis of chronic diffuse glomerular nephritis of two years' standing. There was evidence of definite impairment of the renal function. The eyelids were edematous. The blood pressure was 170 systolic and 128 diastolic, and the hemoglobin content was 70 per cent. Tests for uremia were negative. The urea clearance was 36 per cent of normal. The condition was later diagnosed as polycystic kidney.

Fundic Findings.—The changes of the fundus were limited to a single hemorrhage in one eye.

Case 19 (New York Post-Graduate Medical School and Hospital).—E. G., a man aged 22, was admitted with the general diagnosis of chronic diffuse glomerular nephritis. There was a history of general fatigue and shortness of breath for the past three months. The blood pressure was 284 systolic and 168 diastolic, and the hemoglobin content was 75 per cent. The urea nitrogen content of the blood was 116 mg. per hundred cubic centimeters, and the nonprotein nitrogen content, 190 mg. The patient died of uremia two days after admission.

Fundic Findings.—There were edema of the disk and the surrounding area, venous congestion, constriction of the arteries and arteriovenous compression. Additional findings were numerous hemorrhages, pigmentation, whitish areas and white spots around the macula. This fundic picture illustrates a combination of nephritis, hypertension and arteriolar sclerosis (malignant nephrosclerosis—Fahr).

Case 20 (Harlem Hospital).—T. S., a man aged 45, was admitted with the general diagnosis of chronic glomerular nephritis. He gave a history of dyspnea and of edema of the extremities for the past year. His blood pressure was 220 systolic and 140 diastolic. The specific gravity of the urine was 1.006. The urine contained no sugar. The urea nitrogen content of the blood was 42.6 mg. per hundred cubic centimeters, and the creatinine content, 7.6 mg. The patient died of uremia one week after the examination of the fundus.

Fundic Findings.—The principal finding was ischemia of the disk, with complete obliteration of the normal structure. Ischemia had also probably caused almost total obliteration of the blood vessels and their absorption. Additional findings were a white plaque, striae of pigment and retinal detachment.

Case 21 (Manhattan Eye, Ear and Throat Hospital).—G. G., a boy aged 12 years, was admitted with the diagnosis of ethmoiditis and sphenoiditis of the right side. The rhinologic and roentgen examinations gave positive results. Physical examination showed his general condition to be normal. Vision in the right eye was limited to counting fingers at 6 inches (15 cm.); the left eye was normal, externally and internally. An accurate drawing (fig. 2*A*) is presented, which was taken from a photograph of the fundus before operation on the accessory sinuses.

Fundic Findings.—The disk and its surrounding area were markedly edematous, the veins engorged and tortuous and the arteries contracted. There was a star figure in the macular area. As was noted earlier in the paper, this fundic picture is presented as an illustration of a local toxic factor causing vascular retinal spasm. Three weeks after the performance of an external operation on the ethmoid and the sphenoid sinus another photograph of the fundus was taken, a drawing of which is here presented. In this drawing (fig. 2*B*) the disk appears slightly

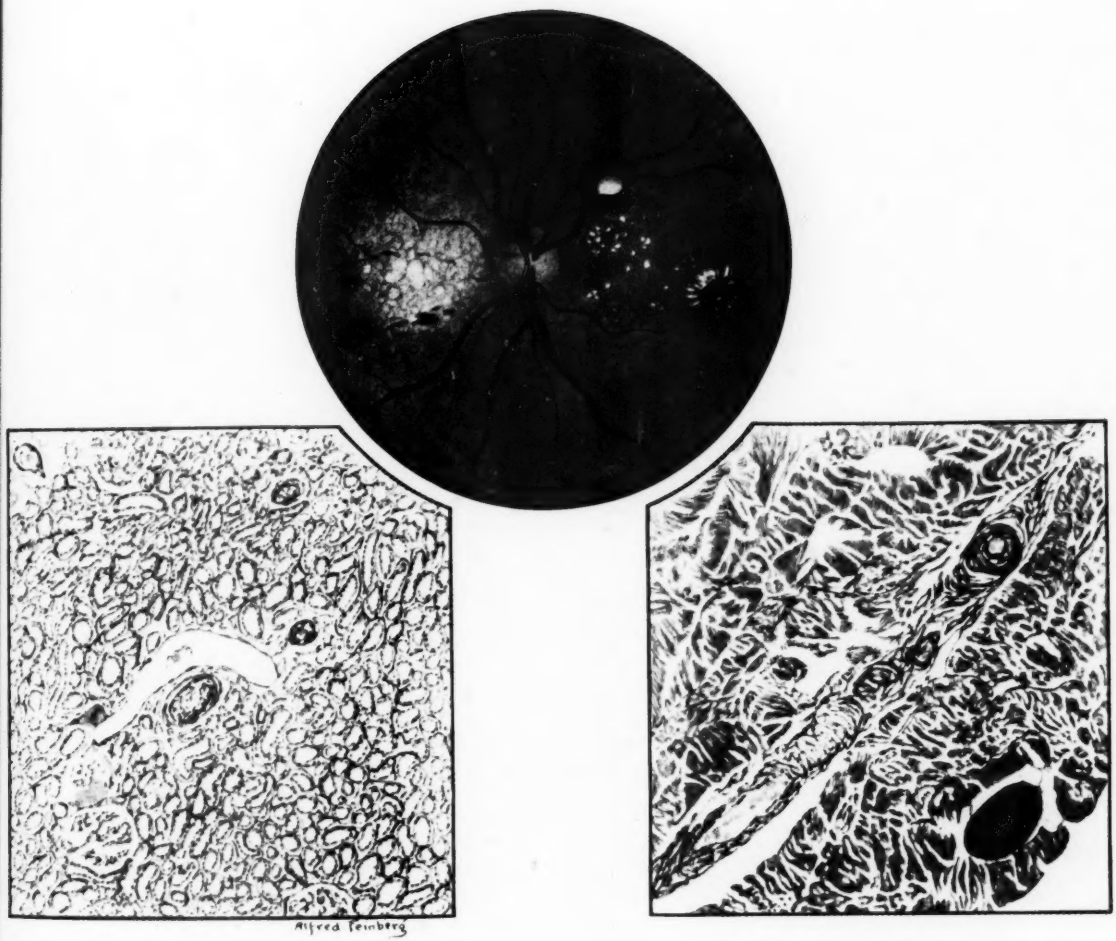

Fig. 1.—The circle shows the appearance of the fundus. The section on the lower left side is a section of the kidney, that on the right, a section of abdominal muscle.

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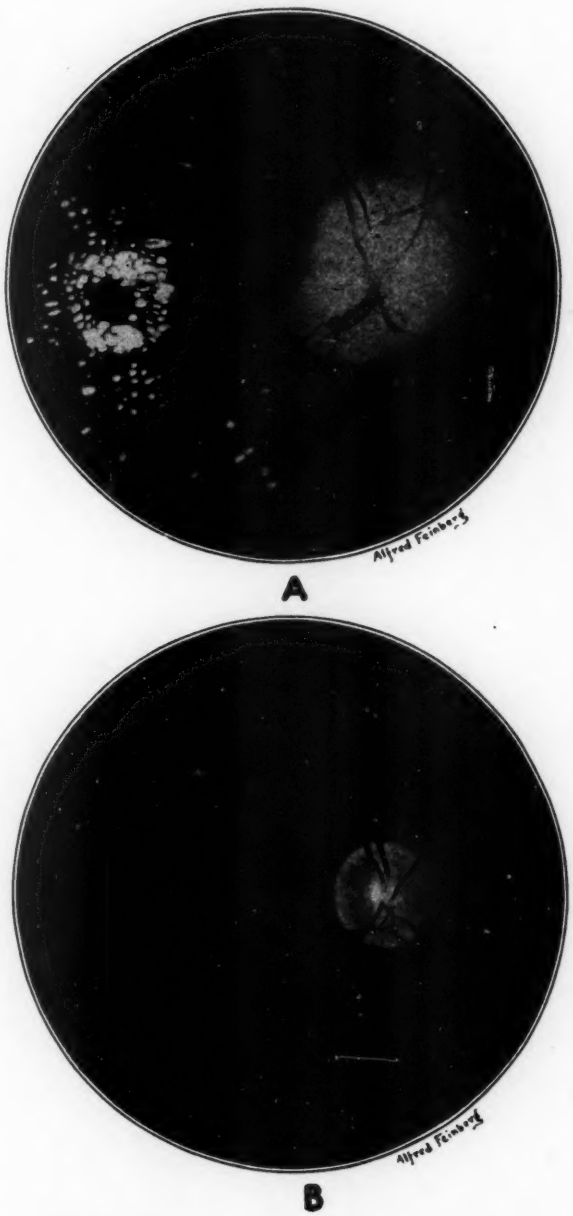

Fig. 2.—*A*, appearance of the fundus before operation on the accessory sinuses. *B*, appearance of the fundus three weeks later.

pale, with a fine diffuse pigmentation in the macular area, and the engorged and dilated veins have resumed a normal appearance and caliber, as may be seen by comparing the two drawings (fig. 2 *A* and *B*).

CASE 22 (New York City Hospital).—B. L., a woman aged 44 years, was admitted with the general diagnosis of nephritis associated with mild cardiac disease. Her blood pressure was 220 systolic and 130 diastolic, and the urea nitrogen content of the blood was 45 mg. per hundred cubic centimeters. Her death was due to cardiac failure.

Fundic Findings.—The disk was edematous, the veins engorged and the arteries contracted. Hemorrhages and glistening white spots could be seen in the macular area.

CASE 23 (Manhattan State Hospital).—E. T., a woman aged 58, was admitted with the general diagnosis of severe diabetes of many years' standing, and hemiplegia and moderate cardiac hypertrophy associated with hypertension. The blood pressure was 230 systolic and 110 diastolic, and the urea nitrogen content was 13 mg. per hundred cubic centimeters. Her death was due to cardiac failure.

Fundic Findings.—The disk was normal but surrounded by white spots. The veins were engorged and tortuous, and there was arteriovenous compression. The presence of hemorrhages was noted.

CASE 24 (New York City Hospital).—G. M., a woman aged 62 years, was admitted with the general diagnosis of senile arteriosclerosis, with superimposed nephritis and cardiac hypertrophy. The blood pressure was 150 systolic and 110 diastolic, and the urea nitrogen content was 138 mg. per hundred cubic centimeters. The patient's death was due to heart failure.

Fundic Findings.—The general findings consisted of moderate sclerosis of the retinal vessels. Microscopic examination of a section of an eye showed moderate edema of the disk. The retina showed cytoid bodies, thickening of the nerve fiber layer and vacuoles. The choroid was normal.

Examination of a section of a kidney revealed sclerosis of the renal vessels and glomerular lesions.

CASE 25 (New York City Hospital).—T. R., a man aged 50, was admitted with the general diagnosis of chronic diffuse glomerular nephritis associated with marked cardiac hypertrophy. The blood pressure was 200 systolic and 120 diastolic, and the urea nitrogen content of the blood was 48 mg. per hundred cubic centimeters. His death was caused by renal and cardiac disease.

Fundic Findings.—The disk was edematous, the veins engorged and indented by sclerotic arterioles and the arteries contracted. There were white spots around the macula, simulating a star in shape.

CASE 26 (New York City Hospital).—H. J., a man aged 40, was admitted with the general diagnosis of essential hypertension (the benign phase). Arteriosclerosis, myocarditis and marked cardiac hypertrophy developed. The blood pressure was 190 systolic and 130 diastolic, and the urea nitrogen content was 17 mg. per hundred cubic centimeters. His death was due to cardiac failure.

Fundic Findings.—The only finding was marked contraction of the retinal arteries. Drawings were made from photomicrographs of a section of an eye showing: moderate edema of the disk (fig. 3 *A*); endarteritis and sclerosis of the retinal vessels, with narrowing of lumen (fig. 3 *B*); sclerosis of arterioles of the choroid, with narrowing of the lumen (fig. 3 *C*).

A drawing of a section of a kidney (fig. 3 *D*) showed sclerosis of arterioles and a fairly well preserved structure. The kidney was not contracted.

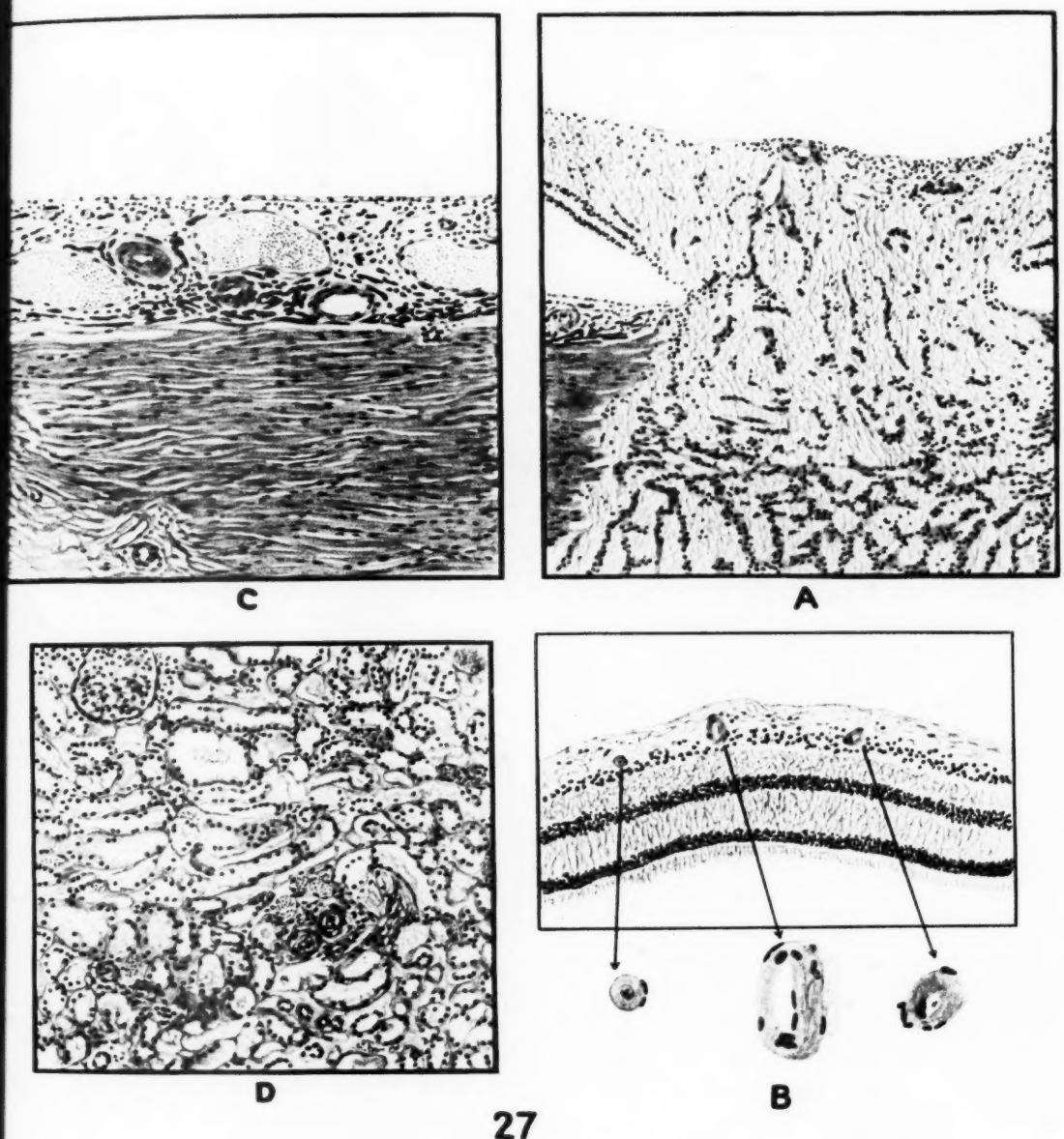

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Fig. 3.—Drawings made from photomicrographs of an eye. *A* illustrates moderate edema of the disk; *B*, endarteritis and sclerosis of the retinal vessels, with narrowing of the lumen, and *C*, sclerosis of arterioles of the choroid, with narrowing of the lumen. *D* is a section of a kidney showing sclerosis of arterioles and fairly well preserved structure.

UNIV. OF MICH.

SUMMARY

A correlation of clinical symptoms with various lesions of the fundus has been presented.

The lesions of the fundus in some of the cases of essential hypertension resembled those seen in nephritis, arterial sclerosis, toxemia of pregnancy and diabetes.

In malignant essential hypertension, however, the lesions of the fundus were more severe than those found in the other diseases considered, with the exception of some types of nephritis. They consisted mainly of marked venous stasis, arterial contraction, profuse hemorrhages, thrombosis, white plaques, macular lesions and edema of the optic disk. These findings were accompanied by normal or practically normal renal function, differentiating malignant essential hypertension from nephritis in a clinical sense.

Hypertension was noted as a common feature in all the cases; accordingly it was difficult, if not impossible, to state on the basis of the fundic findings which condition was primary.

Edema of the optic disk in malignant essential hypertension is significant of its end-stage, which terminates in nephritis, with a grave prognosis regarding the life of the patient.

It is difficult to decide from the lesions of the fundus in essential hypertension when the malignant phase first appeared and what its existing stage is; further study on this question is required.

There may be a similarity between the fundic picture in the terminal stage of malignant essential hypertension and that in chronic glomerulonephritis.

Further microscopic studies of the retinal and choroidal vessels are necessary in order to keep abreast with the more recent investigations of the arterioles of the kidneys and other organs.

The difference in opinion among clinicians regarding the concept of essential hypertension causes various interpretations of the fundic pictures. A uniform clinical concept would establish more firmly a correlation of these fundic pictures with the findings of the internists and pathologists.

The number of cases presented is too small to allow one to draw any definite conclusion at the present time. This report is presented to stimulate further studies and to present drawings of the fundus of patients affected with vascular diseases, which at present is an important field of research.

Mr. Alfred Feinberg made the drawings of the various lesions presented in this paper.

A NEW METHOD FOR REBUILDING A LOWER LID

REPORT OF A CASE

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A large tumor involving the lids always presents two problems: (1) the treatment of the malignant process and (2) repair of any remaining defect for the preservation of the appearance and function of the lid and the protection of the eye. A number of malignant lesions can, of course, be treated by radium and the roentgen rays, and many can be treated by removing surgically only a portion of the lid. When more than half of the lid is involved and it becomes necessary to remove the greater portion or all of the lid, a major problem in plastic surgery is presented, viz., how to build a new lower lid that will be approximately normal in appearance and function.

For use when the upper lid is still intact, a satisfactory solution has been devised, as shown by the following case report and the illustrations.

REPORT OF CASE

History.—Mr. J. N., aged 64, gave a history of having had a small pimple (?) on the left lower lid for seven years, which he had squeezed at times, obtaining a small amount of discharge from it. In the past few months the edge of the lid had turned in, irritating the eye.

Examination.—This revealed a growth (fig. 1) involving the major portion of the left lower lid, extending from the punctum to within 2 mm. of the lateral canthus, producing entropion. It was not adherent to bone, but the skin, tarsus and conjunctiva were all involved. Surgical excision of the mass would mean removal of the entire lower lid. This presented two major problems: how to preserve the eyeball and how to reconstruct a useful and normal-appearing lower lid.

Procedure.—The following operation was devised for this purpose and was satisfactory in respect to both the points mentioned.

First Stage: The entire lower lid was removed along lines of dissection represented by the dotted lines in figure 2, the skin and subcutaneous tissues being severed well below the margin of the mass and the conjunctiva in the lower fornix. Laterally the incision was carried almost to the canthus and medially just beyond the punctum lacrimalis, which was involved. This left a large defect in which a new lower lid was constructed. The skin of the cheek was undermined (figures 2 and 3) sufficiently to allow it to be brought upward to the level normally occupied by the lower lid, without tension. The upper lid was split transversely into two layers, the operator starting along the intermarginal white line

and dissecting upward to a point even with the upper fornix about 3 mm. above the upper margin of the tarsus, not disturbing the attachment of the levator to its upper border. Considerable care must be taken to avoid the roots of the lashes in this dissection.[1] The inner layer was comprised of the tarsus with its conjunctiva. The outer layer was the skin and subcutaneous tissue of the upper lid, with the lashes attached. The lower epithelial border of the upper tarsus

Fig. 1.—Stereophotograph showing the original condition. The growth was a basal cell carcinoma of the lower lid.

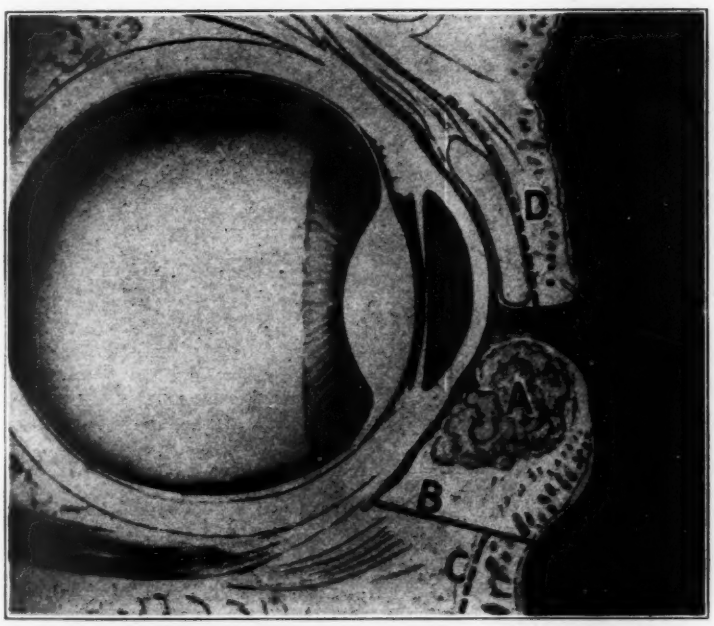

Fig. 2.—Diagrammatic cross-section showing: *A*, the growth; *B*, the line of dissection for removal; *C*, the undermining of the skin to be brought upward, and *D*, the line of dissection in the upper lid.

1. In one case in which operation was performed later, the lashes of the upper lid were lost, and it was necessary to transplant new lashes for both the upper and the lower lid.

was cut off, and this edge was united to the cut conjunctival margin in the lower fornix by fine black silk sutures (000 black silk), the sutures being tied so the knots were on the conjunctival surface. The tarsus in the new position, with the levator attached to its upper edge, is shown diagrammatically in cross-section in figures 3, 4, 5 and 6. The previously undermined skin below was drawn upward and attached to the anterior surface of the lower half of the tarsus by means of three double-armed black silk sutures (fig. 4); so the upper border was midway between the upper and the lower border of the tarsus. The

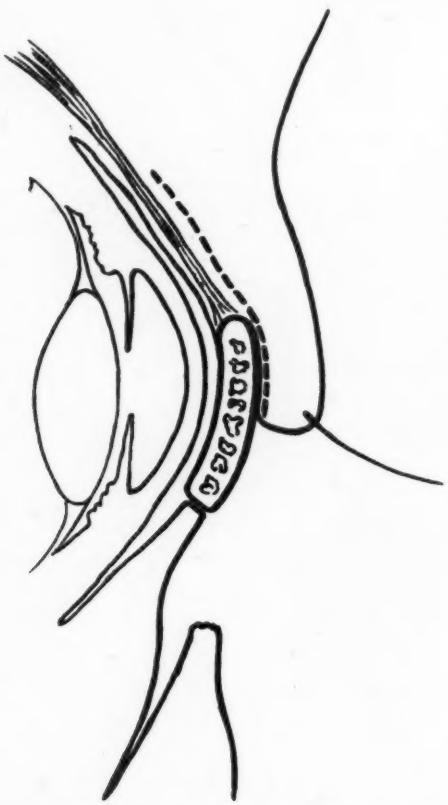

Fig. 3.—Cross-section showing the tarsus pulled down and the conjunctiva pulled up to complete the inner layer.

superficial layer of the upper lid was then attached to the anterior surface of the upper half of the tarsus in the same manner, so that the lashes occupied a transverse straight line parallel and adjacent to the lower edge of the skin across the midportion of the tarsus. The two edges of skin were alined by a subcuticular stitch, and the external portion of the lashes was held upward to the skin of the upper lid by collodion so that the roots of the lashes would take the proper direction. Rubber tissue was put over the area, and a pressure dressing was applied and left in place for six days. Subsequent dressings were applied at intervals of five days for three weeks.

Second Stage: At the end of six weeks transplantation of lashes was done. A transverse trough was dissected down to the tarsus immediately below and parallel to the lashes of the upper lid (fig. 5). A strip of hairs to fit this trough was cut from the lower nasal portion of the opposite eyebrow and, its position being reversed end to end, was laid in the trough and sewn in position (fig. 6). The hairs were then held down by collodion to the skin of the lower lid to cause them to start growing in the proper direction. Rubber tissue was put over the area. A pressure dressing was applied and left in position one week. The dressing was changed again on the twelfth day and removed on the seventeenth day.

The lashes in this strip did not fall out,[2] but kept on growing. The tarsal plate made an excellent smooth base, and the additional raw tissues of the sides

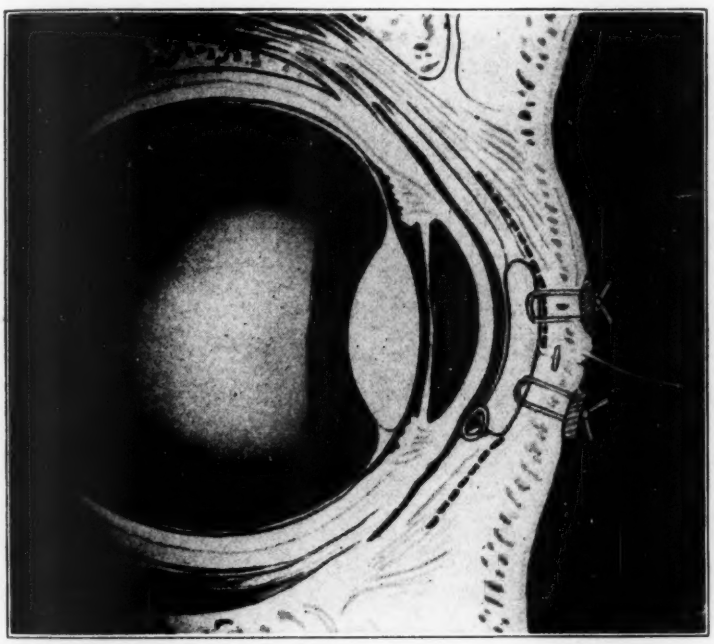

Fig. 4.—Cross-section showing the final arrangement of the upper lid with the skin of the cheek pulled up and attached to the anterior surface of the lower half of the tarsus of the upper lid.

of the trough formed by the dissection supplied ample mechanical and nutritional support for the graft. The proper direction of the lashes was accomplished, first, by turning the transplant so that the portion that was nasal in the right eyebrow was placed nasally in the left lower lid and, second, by anchoring the hairs protruding from the graft to the skin forming the lower lid with collodion in the proper direction (fig. 5). The sutures, which were placed very close to the edges of the skin and tied tightly, were picked off at the first and second dressings, one and two weeks later, respectively.

2. The lashes frequently fall out and later grow in again in the course of two or three months.

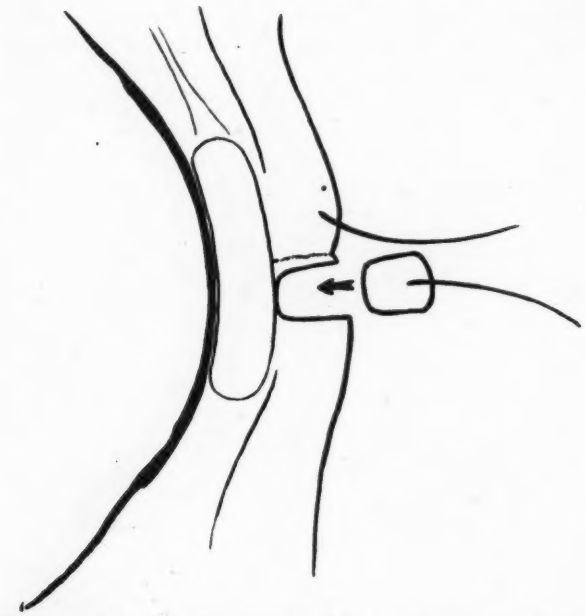

Fig. 5.—Diagram showing the trough dissected down to the tarsus to receive the reversed transplant from the lower portion of the opposite eyebrow.

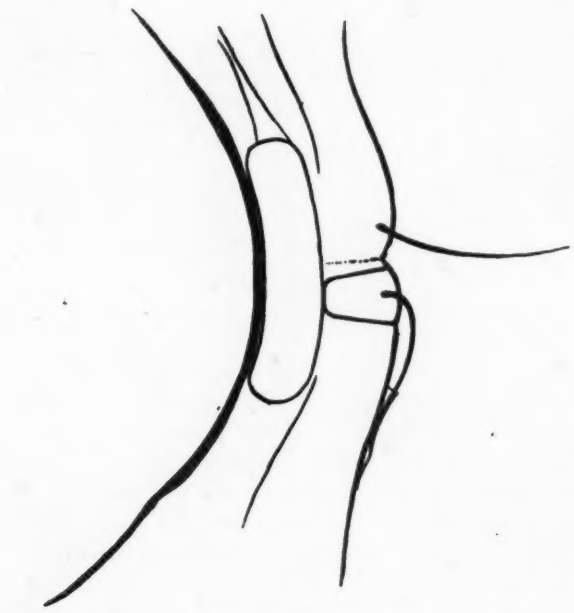

Fig. 6.—Diagram showing the transplant in place. The lashes are held down to skin by collodion.

Fig. 7.—Stereophotograph showing transplanted lashes in position before a transverse opening is made between the two rows of lashes.

Fig. 8.—Photographs taken one month after the final operation. *A* shows the eyes open; *B*, the eyes closed.

Fig. 9.—Photograph showing the lower fornix and the transplanted tarsus, with its conjunctiva, in the lower lid.

Third Stage: After another seven weeks, on March 19, 1936, three months after the original operation, when it was seen that the transplanted lashes of the lower lid were growing in the proper direction (figure 7) and the lashes of the upper lid were complete, an incision was made transversely between the two rows of lashes through the skin and tarsus to open the interpalpebral fissure.

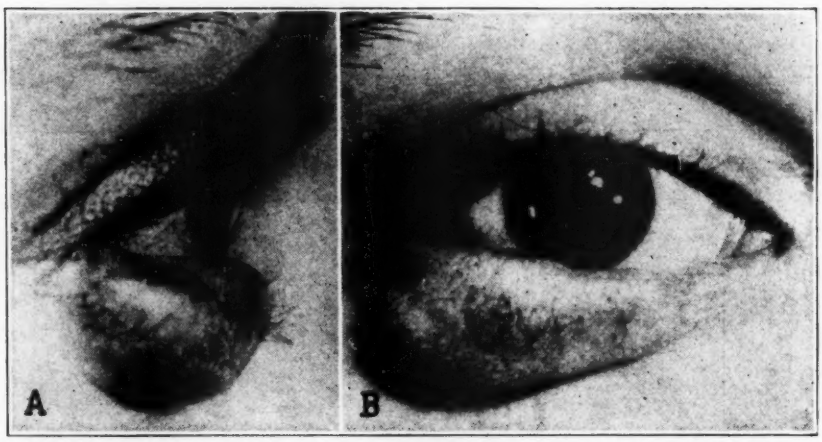

Fig. 10.—*A*, side view of an eye with carcinoma of the meibomian glands of the lower lid; *B*, front view of the eye. The procedure described was carried out.

Fig. 11.—Photograph of an eye with a basal cell carcinoma of the lower lid, on which an operation similar to that described was done.

The new lower lid was now complete except for a punctum and part of the lacrimal canaliculus, which had to be removed because of involvement by the malignant process.

The eye, which was normal in all respects, was preserved intact.

COMMENT

The type of operation described is applicable in the following circumstances:

1. In any case in which a new lower lid structure is necessary, as

2. For repair of a traumatic injury in cases in which the lid structures have been destroyed or mutilated (fig. 13).

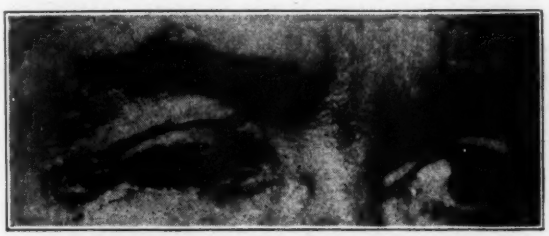

Fig. 12.—Photograph showing a basal cell carcinoma eroding the lower lid and the outer and inner portions of the upper lid, besides an area temporally. In this case the operation described was done.

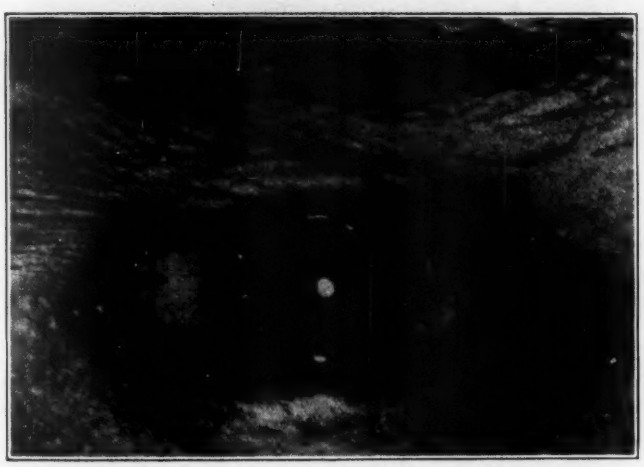

Fig. 13.—Photograph of an eye with chronic blepharitis and deformity of the lids, with loss of lashes from a burn when the patient was 5 years of age. The diseased margins were removed; the upper tarsus was pulled down, and the lashes were transplanted for both lids in the manner described.

3. For reconstruction of the margins of the lid in cases of chronic degenerative conditions in which there is deformity or destruction of the lashes and lid structure (fig. 13) and new lashes are necessary for the upper and lower lids, as in severe cases of ulcerative blepharitis. The resulting lashes are better than when transplanted directly to the margin of the lid.

4. For reconstruction of a portion of the lower lid (fig. 14). When one half or more of the lid must be reconstructed, this procedure becomes the operation of choice, only the temporal or the nasal portion of the upper tarsus being utilized.

5. For reconstruction of an upper lid, the lower lid being used in the same manner as the upper is now used, with an additional insertion of a thin piece of cartilage or tarsus from the opposite upper lid to furnish the rigidity normally furnished by the tarsus. There would be insufficient tarsus in the lower lid for both lids.

Transplantation of the tarsus, with its conjunctiva, from the fellow eye would be feasible when structures are not readily available in the ipsilateral lids.

ADVANTAGES

1. No additional scars are made in the process of the reconstruction.
2. In the new lid most of the normal structures are represented.

Fig. 14.—Photograph showing symmetrical bilateral erosion of the outer half of the lower lids due to infection with Staphylococcus and Streptococcus. In this case the outer half of the upper tarsus was used in the manner described.

3. The function and appearance of the normal lid are fairly well imitated.
4. The margins of the lids close during sleep.
5. The action of the upper lid is not hampered.

SUMMARY

A case is reported in which an epithelioma was present, involving the entire lower lid, which necessitated its complete removal. A new method of reconstruction of the lower lid was used, the cosmetic and functional results being satisfactory. The upper lid was split into two layers transversely. The inner layer, composed of the tarsus with the levator attached to its upper border, was pulled down and was attached to the conjunctiva in the lower fornix. The skin of the cheek was under-

mined, pulled up and united to the anterior surface of the tarsus so that its upper border occupied a transverse position midway between the upper and the lower border of the tarsus. This gave a complete closure of the conjunctival sac except for the medial canthus.

One month later a row of hairs was transplanted from the opposite eyebrow to the anterior surface of the tarsus just below and parallel to the upper row of lashes. A pressure dressing was applied and not disturbed for a week.

Three months after the original procedure a transverse incision was made between the two rows of lashes. A complete new lower lid was then present.

In résumé, (1) the skin was undermined and drawn up from the cheek; (2) the tarsus, with its conjunctiva, was drawn down from the upper lid; (3) the lashes from the opposite eyebrow were transplanted. The eye, which was normal, was at no time endangered.

Twelve operations of this type have been performed to date.

CONCLUSIONS

A method of rebuilding a new lid, when it becomes necessary to remove the existing one or when the existing one is so badly deformed that it needs reconstruction, has been demonstrated. Normal lid structures are used. When necessary, hairs have been transplanted to provide lashes for both the upper and the lower lid in the same manner.[2] The lashes appear not unlike normal ones because of the smooth, firm bed provided by the tarsus and because their growth in the proper direction can be maintained during the period when the graft is taking.

A movie illustrating this process will be lent to responsible persons. There will be a charge of $5 for handling and express.

2. See also 1. When lashes are lacking on the margin of both the upper and the lower lid, as in the case illustrated in figure 14, they may be transplanted into the margin of both the upper and the lower lid.

NAEVUS FLAMMEUS ASSOCIATED WITH GLAUCOMA

REPORT OF A CASE

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Naevus flammeus associated with glaucoma has been recognized since 1860, when Schirmer [1] reported a case of nevus of the face and buphthalmus. Since then about sixty-five cases have been reported, and in these fifteen eyeballs have been examined histologically.

By naevus flammeus, or port wine nevus, is meant a congenital anomaly manifesting itself as a purple discoloration of more or less extensive areas of the skin, the anatomic basis of which consists of telangiectatic vessels. This malformation is more common on the face than on the body and is most often confined to one side of the face, with the median line as its boundary. Not infrequently a striking agreement may be observed between the nevus and the distribution of one or more branches of the trigeminal nerve.

The glaucoma which is associated with this condition is chronic and noninflammatory or primary. The age at which the ocular hypertension occurs will determine whether there is an increase in the size of the globe (buphthalmus). O'Brien and Porter's [2] excellent monograph on this condition showed that the glaucoma was most commonly of the infantile type (in about 68 per cent of the cases). In the majority of cases the glaucoma and nevus are unilateral. Only eight cases of bilateral nevus have been reported, and among these the glaucoma was bilateral in only five.

That naevus flammeus without glaucoma may occur is obvious, since a nevus of the face is fairly common, while its association with glaucoma is rare. Thus it is apparent that glaucoma is associated with facial nevus only in cases in which the lids and other ocular structures are involved. It would be interesting to know whether any cases have been observed in which there is ocular involvement by the nevus without glaucoma.

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1. Schirmer, R.: Ein Fall von Telangiektasie, Arch. f. Ophth. **7**:119, 1860.

2. O'Brien, C. S., and Porter, W. C.: Glaucoma and Naevus Flammeus, Arch. Ophth. **9**:715-728 (May) 1933.

Some of the common ocular findings are: changes in the iris, such as a dense stroma, heterochromia or dilated vessels; changes in the choroid, such as an angioma or nevus; dilated conjunctival vessels; nevus of the conjunctiva and episclera, and varicosities or tortuous retinal vessels, as well as glaucoma.

Histologic examination of those eyes removed (fifteen) showed an angioma of the choroid (eleven eyes), an irregularity of Schlemm's canal and the scleral spur (one eye) and angiomatous changes in the iris and the filtration angle (one eye). The two remaining eyes showed no angioma of the choroid or obstruction of the filtration angle. One eye examined showed bone formation in the choroidal nevus.

Therefore the cause of the glaucoma in the majority of cases may be considered to be due to obstruction of the filtration angle or a choroidal angioma, if the eyes examined can be considered as a cross-section of all eyes with naevus flammeus associated with glaucoma. The etiology of the glaucoma in those eyes in which the filtration angle and the choroid are normal must be due to another factor. Tyson [3] suggested as the etiologic factor the formation of a plasmoid aqueous which obstructs the angle and increases the intra-ocular tension. Increased permeability of the capillaries of the eye may account for a plasmoid aqueous. This was shown by the fluorescin test, which showed a greenish color of the aqueous of the involved eye while the aqueous of the normal eye remained clear.[3] Dunphy [4] gave 50 cc. of a 50 per cent solution of dextrose without effect on the intra-ocular tension, while in a normal eye the tension was reduced by increasing the osmotic pressure of the blood. It would seem, then, that if the protein content of the aqueous of an eye in this group could be obtained some information might be gained. Puncture of the anterior chamber was done on the left eye in the case reported, and a specimen of aqueous was obtained. A protein content of 0.032 per cent, as compared with a normal content of 0.02 per cent, was obtained. But because of the small amount of aqueous available for the test and the technical difficulties experienced, no definite conclusions can be drawn from this result. The protein content of the blood serum of the patient was normal.

It is well known that intracranial changes occur in cases of naevus flammeus.[1] In seven cases there has been a history of epileptiform seizures on the side opposite the facial nevus. In three cases vascular changes were observed in the meninges at autopsy. In five cases calcified meningeal vessels were shown by roentgenograms, and in eight cases there was hemiplegia of the body on the side opposite the nevus.

3. Tyson, H. H.: Naevus Flammeus with Glaucoma, Arch. Ophth. **8**:365-371 (Sept.) 1932.

4. Dunphy, E. B.: Glaucoma Accompanying Nevus Flammeus, Tr. Am. Ophth. Soc. **32**:143-152, 1934.

REPORT OF CASE

L. W., a white man aged 26, was admitted to the hospital on June 1, 1936, with a history of blindness in the left eye for six years; however, this eye had never been normal. About ten months before examination dimness of vision was noted in the right eye, with a blindspot near the center of fixation. There had been no pain in either eye at any time. The occurrence of halos about lights was indefinite. The patient's local physician had diagnosed the condition as chronic

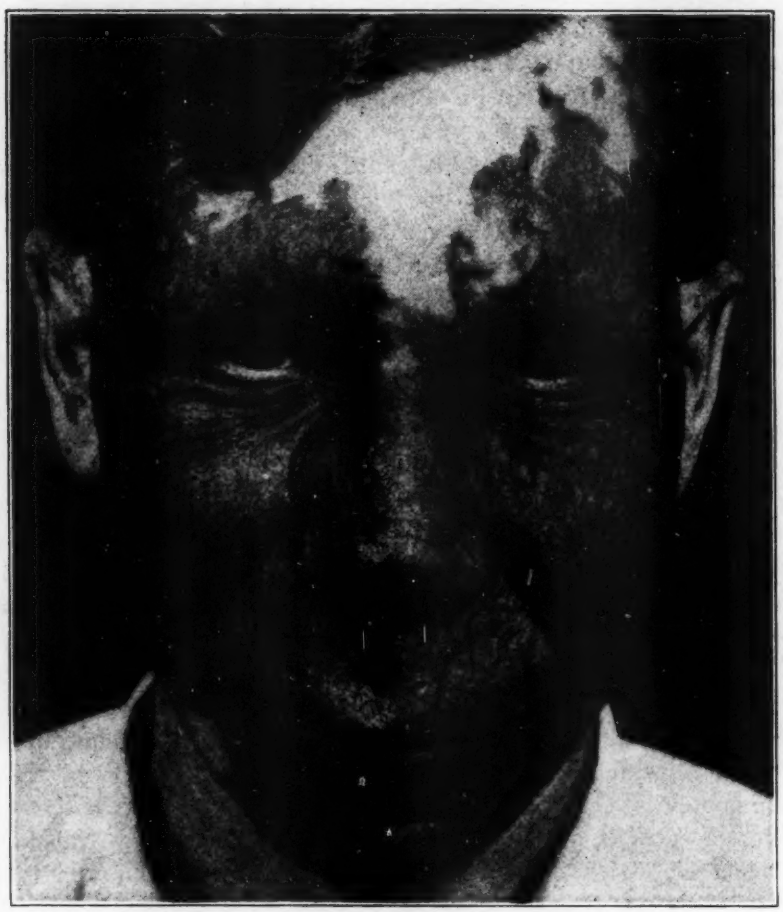

Fig. 1.—Photograph of the nevus, showing its extent.

noninflammatory glaucoma and had given the patient a solution of pilocarpine to be instilled four times a day into each eye. The patient had not used it regularly.

The family history and the past history were not significant. There were nine members in the family, and none of the others had congenital anomalies or birth marks.

Physical examination gave entirely negative results except for a nevus of nearly the whole face. This showed as a deep wine-colored area extending from 2 to 3 cm. above the eyebrows to the chin and laterally to the ears and into the hair at the temples (fig. 1). The mucous membrane of the mouth and the anterior half of the palate were also involved.

Ophthalmologic examination showed that vision was 6/30 —1 in the right eye and nil in the left. With a +75 sphere ⌒ +0.25 cylinder, axis 102, vision of the right eye improved to 6/12—1. Consensual light reflexes were absent. Direct and accommodative reflexes were present in the right eye and absent in the left. In the primary position the left eye diverged 15 degrees according to the Priestly Smith measurement. Extra-ocular movements were normal. The conjunctiva of the lids and globes appeared injected, but on slit lamp examination the vessels of the conjunctiva were seen to be dilated and tortuous. Each cornea appeared to be grossly normal, although measuring 13 mm. in diameter. The anterior chambers were deep, and the irides appeared normal, even on slit lamp

Fig. 2.—Drawing of the fundus of the left eye, showing the tortuosity and anomalous intercommunications of the veins. The disk shows glaucomatous cupping with atrophy.

examination. The pupils were regular in outline and slightly dilated. No evidence of inflammation was present in the anterior segment of either eye. Examination of the fundus of the right eye showed the media to be clear. There was a definite deep glaucomatous cupping of the disk, which was slightly paler than normal. The retina was normal throughout. The veins of the retina were tortuous and anomalous, there being communications between them, as shown in figure 2. The arteries were normal. No hemangiomas of the choroid were seen. Examination of the fundus of the left eye showed a picture identical with that of the fundus of the right eye, except that the glaucomatous cupping was slightly greater and the disk paler. The intra-ocular tension was 35 mm. (Schiötz) in the right eye and 41 mm. in the left. The visual field of the right eye was normal for form and slightly constricted for color, a Bjerrum scotoma being

present (fig. 3). A diagnosis of naevus flammeus associated with chronic non-inflammatory glaucoma was made.

The patient was given a 2 per cent solution of pilocarpine to be instilled four times a day into each eye. Twenty-four hours later the tension was 27 mm. (Schiötz) in the right eye and 35 mm. in the left. Forty-eight hours later, with the same treatment the tension had increased to 42 mm. in the right eye and 48 mm. in the left. Since the tension was not remaining constant, a trephine operation on the right eye was advised; this was performed the following day. When the corneoscleral trephining had been done, peripheral iridectomy performed and the aqueous allowed to escape, the tension of the eye remained hard, and a choroidal hemorrhage was suspected. Examination at the first

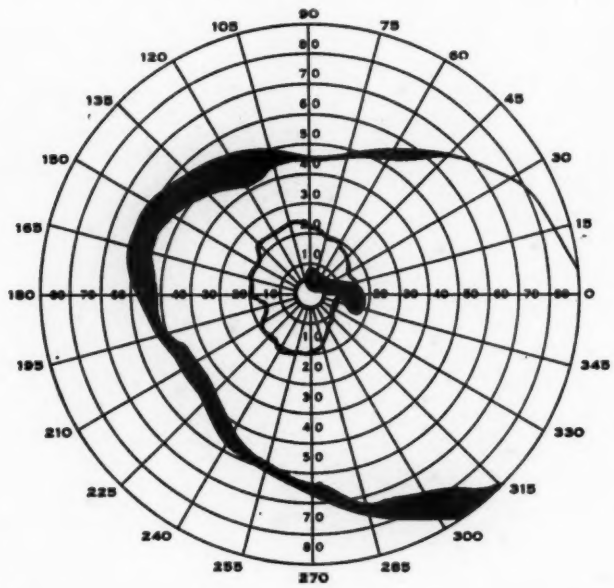

Fig. 3.—Visual field of the right eye, taken on June 3, 1936, with an illumination of 7 candles. Vision was 6/30 + 1. The field for color is limited to the area enclosed by the narrow black line. Colors other than red were not seen.

dressing, however, revealed a complete retinal detachment, the retina appearing to be nearly against the posterior surface of the lens. Gradually the tension became normal, and the retina receded to a more normal position, but it was still detached. A tear in the retina was noted in the periphery at about 10 o'clock. A diathermy operation for the detachment was performed in the region of the tear one month after the trephine operation. This procedure was unsuccessful, and at the time of discharge from the hospital vision of the right eye was 1/60 and the intra-ocular tension was 12 mm., while the intra-ocular tension of the left eye was 37 mm. (pilocarpine was still being used in the left eye). The patient returned after six weeks, at which time a second diathermy operation for the retinal detachment in the right eye was done. This also was unsuccessful.

SUMMARY

Naevus flammeus associated with glaucoma is a rare condition. It was described first by Schirmer,[1] in 1860. It is usually confined to one eye and one side of the face. Naevus flammeus is a congenital anomaly of the face, consisting of telangiectatic vessels. Histologic examination of fifteen eyes removed showed the cause of the glaucoma in thirteen to be due to a choroidal nevus or involvement of the iris, causing obstruction of the filtration angle. In the other two eyes no cause for the glaucoma was found, and it was suspected that in these cases, as well as other cases reported (including the case reported in this paper), the hypertension was due to a plasmoid aqueous caused by increased capillary permeability. Intracranial hemangiomas are found associated with naevus flammeus. Operative procedures on such anomalous eyes are often unsatisfactory, and serious accidents are met with in treating them. (Dunphy [4] did a trephining in one of his cases, and a severe hemorrhage occurred, necessitating removal of the eye.)

SIGNIFICANCE OF RETINAL FATIGUE IN THE STUDY OF GENERAL AND NERVOUS DISEASES

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Within the past few years a study of the fundus oculi has become more important in its relation to the condition of the vascular system. It has been of great value for a longer period as a diagnostic and prognostic aid in many conditions affecting the general health of the patient. The purpose of our present study is to utilize the condition of the retina, perhaps the most highly specialized nerve tissue in the body, as a possible index in connection with the condition of the general nervous system of the patient.

In this connection a simple test was devised to determine the fatigability of the retina. This test in its simplest form requires little time and only a fair amount of intelligence on the part of the patient. A small black dot or circle is placed at the center of a piece of white paper. The patient, with one eye occluded, is instructed to look steadily at the dot or circle, without winking or changing his gaze, until a wave of darkness or of mottling passes over the whiteness of the paper. This period is usually only a few seconds and may be taken as the time required for the retina of the eye used to become fatigued (the fatigue time). If the patient is directed to continue gazing, it will be found that the wave of darkness passes and is followed in a few seconds by another. This may be continued, to demonsrtate that the waves occur in regular cycles. The retina, therefore, must be considered as a delicate nervous membrane which at best is fatigued easily and quickly.

It will be found that this fatigue time varies with the intensity of the light used, and there is variation when different colors are used. In our studies an attempt was made to standardize and control these and other factors. The patient was seated in front of a small table which was covered in a neutral shade of green. Testing cards of the same shade were used. Mounted on each of these cards was a square of one of the standard Heidelberg papers. Five colors were used: red, yellow,

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blue, green and white. In the center of each of these squares a small black dot was placed. The card to be used was placed on the table, illuminated by a daylight lamp so that the intensity of the light on the card was 25 foot-candles. The patient's eyes were kept at a distance of 18 inches (45.7 cm.) from the card. A stopwatch was used, and the fatigue time was determined in each instance as the number of seconds between the time when the gaze was directed to the dot at the center of the card and the time at which the wave of darkness or of mottling seemed to pass over the card. All the tests were conducted by the same operator. Many had to be repeated because of faulty cooperation on the part of the patient. If the eye was moved or the lids winked the patient was allowed to rest and the test was repeated. The cards were presented in the same order to each patient: first, red, then yellow, then blue, then green and last white. In each case the left eye was tested first.

A distinction was made between those patients with eyes the refractive error of which was sufficient to warrant the use of glasses (the term "corrected vision" is used to designate such a condition in the tables) and those whose refractive error was negligible (the term "normal vision" is used in the tables). Also, a distinction was made between patients with light eyes and those with dark eyes. The group tested included two hundred and five patients whose age ranged from 15 to 68 years, the average age being 43.6 years.

In testing retinal fatigue for eyes with normal vision and those with corrected vision (table 1) it was found that the retina of the average eye became fatigued most quickly when the patient looked at the red card. The colors next in order in producing fatigue were yellow, then blue, then white and last green. The retina of the average eye with normal vision became fatigued less quickly than the retina of the average eye with corrected vision. No definite conclusion was drawn in regard to the difference between the retinas of light eyes and those of dark eyes.

In testing retinal fatigue in cases of ocular conditions (table 2) the retinas of the eyes showing retinal arteriosclerosis tired most quickly. The retina of the average eye with hyperopia or hyperopic astigmatism became fatigued more quickly than that of the eye with myopia or myopic astigmatism, and the retina of the latter became fatigued more quickly than that of the average eye with normal vision.

The group of persons with psychoneurosis (table 3) formed 21.7 per cent of the total group. The retina of the average patient with psychoneurosis became fatigued much more quickly than that of the average patient.

Both malnutrition and tuberculosis appeared to play a part in retinal fatigue (table 4). However, the retinas of the average patient with

TABLE 1.—*Retinal Fatigue for Eyes with Normal Vision and Eyes with Corrected Vision* *

	Number of Cases	Percentage of Cases	Total Range, Seconds	Red Average Fatigue Time, Seconds		Yellow Average Fatigue Time, Seconds		Blue Average Fatigue Time, Seconds		Green Average Fatigue Time, Seconds		White Average Fatigue Time, Seconds	
				OS	OD	OS	OD	OS	OD	OS	OD	OS	OD
Normal vision	75	37.2	1-125	4.9	4.7	8.6	8.3	8.4	8.7	9.2	11.1	8.9	11.6
Corrected vision	127	62.8	0-55	4.7	4.4	6.9	6.5	7.8	7.7	8.5	8.2	7.9	7.9
Total number of cases of normal and of corrected vision	202	100.0	0-125	4.8	4.5	7.7	7.4	8.1	8.2	8.2	9.6	8.4	9.7
Dark eyes with normal vision	31	15.3	1-67	4.1	4.1	8.8	8.3	9.0	9.7	9.0	12.0	10.3	10.1
Light eyes with normal vision	44	21.8	1-125	4.4	3.7	7.5	8.3	8.5	8.1	8.4	10.2	8.3	8.4
Dark eyes with corrected vision	48	23.8	1-55	5.0	4.2	7.0	6.2	8.5	7.8	7.8	8.6	8 2	7.9
Light eyes with corrected vision	79	39.1	0-35	4.8	4.7	6.8	6.8	7.1	7.7	9.0	7.9	7.4	7.7

* Of the total group, 60.9 per cent had light eyes, and 39.1 per cent had dark eyes.

TABLE 2.—*Retinal Fatigue in Cases of Ocular Conditions*

	Number of Cases	Percentage of Cases	Total Range, Seconds	Red Average Fatigue Time, Seconds		Yellow Average Fatigue Time, Seconds		Blue Average Fatigue Time, Seconds		Green Average Fatigue Time, Seconds		White Average Fatigue Time, Seconds	
				OS	OD	OS	OD	OS	OD	OS	OD	OS	OD
Normal vision	75	37.20	1-125	4.9	4.7	8.6	8.3	8.4	8.7	9.2	11.1	8.9	11.6
Corrected vision	127	62 80	0-55	4.7	4.4	6.9	6.5	7.8	7.7	8.5	8 2	7.9	7.9
Total number of cases of normal and of corrected vision	202	100.00	0-125	4.8	4.5	7.7	7.4	8.1	8.2	8.8	9.6	8.4	9.7
Myopia and myopic astigmatism	29	14.35	1-31	4.4	4.9	6.5	6.0	7.2	6.6	8.0	6.7	7.1	6.3
Hyperopia and hyperopic astigmatism	59	29.21	1-28	3.6	3.0	5.1	5.5	6.0	6.3	6.6	6.7	6.5	6 2
Divergent strabismus	4	1.98	2-4	4.0	3.7	4.8	6.5	6.3	8.2	6.2	8.3	6.2	8.5
Amblyopia ex anopsia	8	3.96	1-16	4.7	4.0	8.0	5.3	5.8	5.6	7.8	7.2	6.5	6.2
Retinal arteriosclerosis	3	1.48	2-8	2.6	2.8	4.6	4.0	5.0	5.3	4.5	4.6	6.3	4.6
Bilateral conjunctivitis	5	2.47	2-55	3.2	3.9	5.6	6.2	7.8	10.5	7.4	8.2	5.3	9 0
Other ocular conditions*	12	5.89	1-28	4.8	3.4	7.1	6.6	5.9	5.7	7.9	8.1	8.9	6.0
Total number of ocular conditions	120	59.40	1-31	4.0	3.7	6.0	5.8	6.4	6.6	7.4	5.4	7.0	6.5

* These were retinitis pigmentosa (single cases), possible retrobulbar neuritis, papilledema, lesion of the right supra-orbital nerve, multiple corneal ulcers, partial bilateral atrophy of the optic nerve, a slight corneal scar in the left eye, detachment of the retina in the right eye, uveitis in the right eye, atrophic bilateral choroiditis and mild retinal asthenopia.

TABLE 3.—*Retinal Fatigue in Cases of Neuropsychiatric Conditions*

	Number of Cases	Percentage of Cases	Total Range, Seconds	Red Average Fatigue Time, Seconds		Yellow Average Fatigue Time, Seconds		Blue Average Fatigue Time, Seconds		Green Average Fatigue Time, Seconds		White Average Fatigue Time, Seconds	
				OS	OD	OS	OD	OS	OD	OS	OD	OS	OD
Normal vision	75	37.2	1-125	4.9	4.7	8.6	8.3	8.4	8.7	9.2	11.1	8.9	11.6
Corrected vision	127	62.8	0-55	4.7	4.4	6.9	6.5	7.8	7.7	8.5	8.2	7.9	7.9
Total number of cases of normal and of corrected vision	202	100.0	0-125	4.8	4.5	7.7	7.4	8.1	8.2	8.8	9.6	8.4	9.7
Psychoneurosis associated with normal vision	18	8.9	1-43	3.0	2 8	5.0	6.2	5.8	7.2	7.1	9.4	8.0	7.6
Psychoneurosis associated with corrected vision	26	12.8	0-25	2.5	2.6	5.7	4.4	5.7	5.7	7.0	5.3	5.3	5.4
Total number of cases of psychoneurosis associated with normal vision and with corrected vision	44	21.7	0-43	2.9	2.7	5.4	5.1	5.7	6.3	7.0	6.9	6.4	6.3
Other neuropsychiatric conditions associated with normal vision	17	8.4	3-43	5.0	4.3	8.8	4.7	8.3	6.8	10.0	8.3	10.0	6.7
Other neuropsychiatric conditions associated with corrected vision	20	9.9	2-32	3.8	2.8	5.9	4.4	5.4	5.7	7.2	6.8	7.3	6.9
Total number of other neuropsychiatric conditions	37	18.3	2-43	4.4	3.5	7.2	4.5	6.8	6.2	8.9	7.5	8 9	6.8
Total number of cases of neuropsychiatric conditions	81	40.0	0-43	3.6	3.0	6.2	4.8	6.2	6.2	7.9	7.2	7.5	6.6

TABLE 4.—*Retinal Fatigue in Cases of Malnutrition and Tuberculosis*

	Number of Cases	Percentage of Cases	Total Range, Seconds	Red Average Fatigue Time, Seconds		Yellow Average Fatigue Time, Seconds		Blue Average Fatigue Time, Seconds		Green Average Fatigue Time, Seconds		White Average Fatigue Time, Seconds	
				OS	OD	OS	OD	OS	OD	OS	OD	OS	OD
Normal vision	75	37.20	1-125	4.9	4.7	8.6	8.3	8.4	8.7	9.2	11.1	8.9	11.6
Corrected vision	127	62.80	0-55	4.7	4.4	6.9	6.5	7.8	7.7	8.5	8.2	7.9	7.9
Total number of cases of normal and of corrected vision	202	100.00	0-125	4.8	4.5	7.7	7.4	8.1	8.2	8.8	9.6	8.4	9.7
Malnutrition associated with normal vision	7	3.40	1-32	3.2	2.2	4.7	4.3	7.2	8.6	6.3	6.1	8.5	6.2
Malnutrition associated with corrected vision	15	7.40	1-19	2.8	2.5	5.8	4.0	4.3	5.3	5.6	6.4	5.9	5.7
Total number of cases of malnutrition associated with normal vision and with corrected vision	22	10.80	1-32	3.0	2.4	5.4	4.3	5.5	6.7	5.8	6.6	6.7	5 9
Tuberculosis associated with normal vision	8	3.96	1-25	3.3	2.7	4.5	6.1	7.8	7.0	8.5	10.5	6.6	7.9
Tuberculosis associated with corrected vision	12	5.94	1-16.5	3.9	2.6	5.3	5.4	5.1	6.5	5.4	8.0	6.2	6.8
Total number of cases of tuberculosis associated with normal vision and with corrected vision	20	9.90	1-25	3.7	2.6	5.0	5.7	6.2	6.7	6.7	9.0	6.4	7.2

TABLE 5.—*Retinal Fatigue in Cases of Chronic Tonsillitis, Chronic Sinusitis, Chronic Rhinitis, Pyorrhea and Dental Foci of Infection*

	Number of Cases	Percentage of Cases	Total Range, Seconds	Red Average Fatigue Time, Seconds		Yellow Average Fatigue Time, Seconds		Blue Average Fatigue Time, Seconds		Green Average Fatigue Time, Seconds		White Average Fatigue Time, Seconds	
				OS	OD	OS	OD	OS	OD	OS	OD	OS	OD
Normal vision	75	37.20	1-125	4.9	4.7	8.6	8.3	8.4	8.7	9.2	11.1	8.9	11.6
Corrected vision	127	62.80	0-55	4.7	4.4	6.9	6.5	7.8	7.7	8.5	8.2	7.9	7.9
Total number of cases of normal and of corrected vision	202	100.00	0-125	4.8	4.5	7.7	7.4	8.1	8.2	8.8	9.6	8.4	9.7
Tonsillitis associated with normal vision	16	7.92	1-45	4.1	3.3	6.3	4.6	6.7	6.1	9.3	7.1	9.0	6.2
Tonsillitis associated with corrected vision	22	10.88	1-40	6.2	4.5	6.5	6.7	7.8	7.5	6.5	6.7	7.5	7.2
Total number of cases of tonsillitis associated with normal vision and with corrected vision	38	18.80	1-45	5.2	4.0	6.4	5.9	7.4	6.9	7.7	6.8	8.1	6.8
Sinusitis associated with normal vision	3	1.50	1-13	3.1	3.8	4.0	4.3	3.3	6.6	4.5	5.0	3.3	3.6
Sinusitis associated with corrected vision	9	4.40	1-20	3.6	2.8	6.3	3.6	5.8	4.7	7.0	5.5	5.9	5.1
Total number of cases of sinusitis associated with normal vision and with corrected vision	12	5.90	1-20	3.5	3.0	5.7	3.8	5.2	5.2	6.4	5.3	5.2	4.7
Rhinitis associated with normal vision	4	1.98	1-18	3.2	2.1	4.8	3.8	6.1	6.0	9.2	8.0	8.0	7.7
Rhinitis associated with corrected vision	6	2.97	1-8	2.8	2.8	4.5	3.5	5.0	4.0	4.9	4.8	4.3	4.6
Total number of cases of rhinitis associated with normal vision and with corrected vision	10	4.95	1-18	3.0	2.5	4.6	3.7	5.4	4.8	6.6	6.1	5.8	5.9
Pyorrhea associated with normal vision	15	7.40	1-45	4.0	2.8	5.5	5.3	6.7	7.2	7.5	6.5	8.3	6.6
Pyorrhea associated with corrected vision	33	16.30	1-31	3.7	3.5	5.6	5.3	7.1	7.1	7.0	6.3	6.7	6.2
Total number of cases of pyorrhea associated with normal vision and with corrected vision	48	23.70	1-45	3.8	3.3	5.6	5.3	7.0	7.2	7.1	6.4	7.2	6.3
Dental foci of infection associated with normal vision	12	5.90	1-125	3.1	3.0	4.8	12.4	5.5	8.1	6.7	8.6	5.9	16.3
Dental foci of infection associated with corrected vision	22	10.90	1-55	4.9	5.0	7.7	6.3	10.4	8.1	7.5	7.6	6.9	6.5
Total number of cases of dental foci of infection associated with normal vision and with corrected vision	34	16.80	1-125	4.3	4.3	6.7	8.5	8.7	8.1	7.2	7.9	6.5	10.0

TABLE 6.—*Retinal Fatigue in Cases of Hypertension and Arteriosclerosis*

	Number of Cases	Percentage of Cases	Total Range, Seconds	Red Average Fatigue Time, Seconds		Yellow Average Fatigue Time, Seconds		Blue Average Fatigue Time, Seconds		Green Average Fatigue Time, Seconds		White Average Fatigue Time, Seconds	
				OS	OD	OS	OD	OS	OD	OS	OD	OS	OD
Normal vision	75	37.20	1-125	4.9	4.7	8.6	8.3	8.4	8.7	9.2	11.1	8.9	11.6
Corrected vision	127	62.80	0-55	4.7	.44	6.9	6.5	7.8	7.7	8.5	8.2	7.9	7.9
Total number of cases of normal and of corrected vision	202	100.00	0-125	4.8	4.5	7.7	7.4	8.1	8.2	8.8	9.6	8.4	9.7
Hypertension associated with normal vision	3	1.48	2-27	3.3	3.6	10.3	6.6	11.0	7.5	13.0	10.8	9.3	10.1
Hypertension associated with corrected vision	14	6.93	1-40	7.0	5.5	7.5	8.3	10.0	7.1	8.5	9.4	8.7	8.1
Total number of cases of hypertension associated with normal vision and with corrected vision	17	8.40	1-40	6.4	5.2	7.6	8.0	10.3	7.1	9.3	9.6	8.9	8.4
Arteriosclerosis associated with normal vision	0	....	....	...	...	...	...	...	...	...	...	...	...
Arteriosclerosis associated with corrected vision	12	5.90	1-29	4.5	4.0	7.8	5.8	6.5	5.9	9.1	8.2	7.6	8.3

TABLE 7.—*Retinal Fatigue in Cases of Syphilis, Chronic Alcoholism, Cephalalgia and Migraine*

	Number of Cases	Percentage of Cases	Total Range, Seconds	Red Average Fatigue Time, Seconds		Yellow Average Fatigue Time, Seconds		Blue Average Fatigue Time, Seconds		Green Average Fatigue Time, Seconds		White Average Fatigue Time, Seconds	
				OS	OD	OS	OD	OS	OD	OS	OD	OS	OD
Normal vision	75	37.20	1-125	4.9	4.7	8.6	8.3	8.4	8.7	9.2	11.1	8.9	11.6
Corrected vision	127	62.80	0-55	4.7	4.4	6.9	6.5	7.8	7.7	8.5	8.2	7.9	7.9
Total number of cases of normal and of corrected vision	202	100.00	0-125	4.8	4.5	7.7	7.4	8.1	8.2	8.8	9.6	8.4	9.7
Syphilis associated with normal vision	4	1.98	1.5-28	4.7	2.7	12.0	6.2	12.0	6.6	14.0	8.3	11.2	7.1
Syphilis associated with corrected vision	3	1.48	1-7	2.0	1.8	4.3	3.3	4.0	4.0	4.8	3.8	5.1	3.8
Total number of cases of syphilis associated with normal vision and with corrected vision	7	3.46	1-28	3.5	2.2	8.7	5.0	8.5	5.5	11.5	6.4	8.6	5.7
Chronic alcoholism associated with normal vision	1	0.49	1-8	2.0	1.0	8.0	4.0	3.0	4.0	5.0	5.0	4.0	6.0
Chronic alcoholism associated with corrected vision	4	1.98	1-15	2.5	2.7	4.5	5.1	6.1	7.0	6.7	7.7	5.7	7.2
Total number of cases of chronic alcoholism associated with normal vision and with corrected vision	5	2.47	1-15	2.4	2.4	5.2	4.9	5.5	6.5	6.5	7.2	5.4	7.0
Cephalalgia associated with normal vision	0	....	....	...	...	...	...	...	...	...	...	...	...
Cephalalgia associated with corrected vision	2	0.99	2-29	8.5	3.5	12.5	6.0	19.0	8.0	17.0	7.5	10.0	6.0
Migraine associated with normal vision	0	....	....	...	...	...	...	...	...	...	...	...	...
Migraine associated with corrected vision	1	0.49	1-9	2.0	1.0	4.0	2.5	6.5	5.0	9.0	4.0	7.0	4.0
Total number of cases of cephalalgia and of migraine	3	1.48	1-29	6.3	2.6	9.6	4.8	14.8	7.0	14.3	6.3	9.0	5.3

tuberculosis did not become fatigued quite so quickly as those of the average patient with malnutrition.

The retinas of the patients with the more common foci of infection (table 5) became fatigued more quickly than those of the average patient. In this group the patients with rhinitis showed retinal fatigue most quickly; then, in order, were those with sinusitis, those with pyorrhea, those with tonsillitis and those with dental abscesses.

In the cases studied, the retinas of the average patient with hypertension (table 6) had a longer fatigue time (became fatigued less easily) than those of the average patient. The average patient with arteriosclerosis showed retinal fatigue a little more quickly than the average patient.

The number of patients with syphilis, chronic alcoholism, cephalalgia and migraine (table 7) were too few to enable one to draw definite conclusions, but in cases of each of these conditions the fatigue time was much shortened.

CONCLUSIONS

The condition of the nervous system as indicated by the condition of the retina, one of its most sensitive components, may be studied by means of a simple test for retinal fatigability.

The average retina becomes fatigued most quickly when looking at red; then in order come yellow, blue, white and green.

The retinas of patients with psychoneurosis become fatigued more quickly than those of average patients; and of these, those of patients with neurasthenia show marked fatigability.

The retina becomes fatigued more quickly in cases of malnutrition than in cases of tuberculosis.

Patients with foci of infection show an increased retinal fatigability.

Simple hypertension appears to lessen retinal fatigability, arteriosclerosis to increase it.

In general, a determination of the fatigue time of the retina will be of value in a general examination of patients, especially those with conditions involving the nervous system.

GLAUCOMA AND SYMPATHETIC OPHTHALMIA

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Glaucoma not infrequently comes into consideration in the clinical and pathologic study of sympathetic ophthalmia, as regards both the exciting eye and the sympathizing eye. The complication of glaucoma in the uninjured eye is always a matter of the gravest concern. One naturally tries to dilate the pupil, because sympathetic ophthalmia is only too often characterized by adhesions at the pupillary border. On the other hand, if the tension rises still higher, further use of a mydriatic is contraindicated, while the use of pilocarpine may tend to produce more pupillary adhesions. The difficulty in handling this type of glaucoma becomes exceedingly critical when the tension is very high.

One should bear in mind, in cases of glaucoma associated with sympathetic ophthalmia, that the stroma of the iris is transformed into a specific granulation tissue the slightest handling of which is sure to be followed by an exacerbation of the inflammation. This transformation renders operative procedure on the iris impossible, because one would be dealing not with the stroma of the iris but with a tissue of a far different character.

Neither may an operation be attempted in the region of the ciliary body, because the ciliary body is a place of predilection for the specific infiltration. Cyclodialysis is especially contraindicated, just as it is in every case of secondary glaucoma due to ordinary iridocyclitis, for the spatula presses on the outer surface of the inflamed ciliary muscle, and as it lacerates its insertion to the anterior scleral spur it ruptures blood vessels and causes hemorrhage. The trephining operation of Elliott is contraindicated, owing to the speedy closure of the canal by an accumulation of fibrin and exudate.

Increase in tension in the exciting eye is usually the direct result of the injury, as in cases in which the iris is incarcerated or the lens is swollen. It is hopeless to attempt to reduce the tension in the exciting eye which has already been damaged, once sympathetic ophthalmia develops.

Read at the meeting of the Section of Ophthalmology of the New York Academy of Medicine, Jan. 18, 1937.

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A special complication of the glaucoma in the sympathizing eye is the picture of iris bombé, concerning which an exact understanding is required.[1] In ordinary iritis, seclusion or occlusion of the pupil is the cause of iris bombé, which is an expression of an increase of fluid in the posterior chamber carrying the iris forward and stretching it. Iridectomy or transfixion at two points gives a satisfactory result. In sympathetic ophthalmia the clinical picture of iris bombé is caused only exceptionally by the pressure of fluid in the posterior chamber; it is due generally to an excessive proliferation of the specific granulation tissue that replaces the stroma of the iris, causing its anterior limiting layer to approach the cornea. This sympathetic infiltration, as it develops in the posterior layers of the iris, destroys the pigment epithelium, so that it lies in direct contact with the lens. There is presented a picture of complete posterior synechia, and at the same time the infiltration may even fill the circumlental space. This method of the development and spread of the nodules posteriorly, with destruction of the pigment epithelium, is a familiar observation in preparations of eyes with sympathetic ophthalmia. Clinically, an analogous occurrence may be observed in tuberculosis of the iris, in the form of small grayish nodules that appear along the anterior surface of the pupillary margin. With the slit lamp they may be accurately studied and may be seen occasionally in the act of detaching and floating in the anterior chamber until they are finally deposited on Descemet's membrane. Nodules in the posterior portion of the iris are so common in sympathetic ophthalmia and so rare in other types of iritis as to be considered almost pathognomonic for sympathetic ophthalmia.

It is of special clinical importance to know that in sympathetic ophthalmia the anterior limiting layer of the iris is not destroyed but may retain much of its normal appearance and color in spite of the mass of underlying granulation tissue that has replaced the stroma. Tuberculosis of the iris soon manifests itself by developing forward and destroying the anterior limiting layer of the iris so that naked tuberculous nodules are clinically seen.

SURGICAL PROCEDURE FOR THE REDUCTION OF TENSION IN THE SYMPATHIZING EYE

In cases of increased tension in an eye with sympathetic ophthalmia one should bear in mind that the secondary type of glaucoma is considered less apt to injure the nerve fibers at the papilla than the primary type, even though in the former a tension of 60 or 70 mm. may be reached. In sympathetic ophthalmia the sclera may be so highly inflamed

1. Samuels, B.: Notes on the Pathology and Surgical Treatment of Sympathetic Ophthalmia, Arch. Ophth. **15**:59-69 (Jan.) 1936.

that one speaks of sympathetic scleritis, and the cribriform fascia and the pial sheath of the nerve may be particularly involved.[2] It is thought, on account of the congestion in all parts of an eye with the inflammatory type of glaucoma, that the cribriform fascia withstands pressure better and recedes less than in an eye with the noninflammatory type. Then, too, in an eye with sympathetic ophthalmia the increased tension may not be of long duration, because it is the fate of this process that the tension becomes normal or soft in time.

In cases in which the tension continues to be high and cannot be relieved by medicinal treatment, either local or systemic, paracentesis is in order, the proper site for which is in the cornea, 1 mm. from the engorged blood vessels of the limbus. The paracentesis incision must of necessity be reopened repeatedly. On this account care should be taken to make the incision large. It is also important to note its exact location in order to avoid abrading the epithelium and scratching Bowman's membrane, which tend to increase irritation. Thorough cocainization should precede the reopening, because eyes with this condition are extremely sensitive. The epithelial adhesion should be cautiously broken up with a spatula by directing the instrument in such a way as to depress the peripheral lip of the wound. No form of fixation of the eye should be used because of the pain it would produce. If the original incision is large enough, merely depressing the peripheral lip is sufficient, because by the second day the wound is closed solely by epithelium. The aqueous escapes readily. It is not necessary for the spatula to enter the anterior chamber. It is best to avoid the risk of touching the iris. Finally, the escape of aqueous should be brought about slowly, as a rapid escape is likely to be followed by hemorrhage from the iris.

After three or four days, reopening of a paracentesis incision is no longer possible on account of the permanent healing of the wound. However, the procedure of reducing the tension for a few hours at a time is of great help in sympathetic ophthalmia, relieving congestion in the uvea and retina and tiding the disease over a critical glaucomatous phase, which of itself may not be of long duration.

If paracentesis does not prove successful, one may be driven, as a last resort, to the only other surgical alternative possible, namely, anterior sclerotomy. However, the anterior sclerotomy here referred to differs from the operation ordinarily performed in that the incision must pass through a violently inflamed sclera. Above all, one has to avoid even touching the iris, much less attempt to excise or incarcerate it. Under these circumstances this undertaking is difficult, and as likely

2. Samuels, B.: Sympathetic Scleritis, Tr. Am. Ophth. Soc. **31**:223-231, 1933.

as not will be useless, owing to failure to obtain a fistulous opening. All told, it is evident that the surgical means of relieving glaucoma in an eye with sympathetic ophthalmia are beset with limitations.

As an example of the deleterious effect of operating on the sympathizing eye I cite the case of a man who suffered from sympathetic ophthalmia for thirty years. The injured eye had been enucleated, and the fellow eye retained vision of 10/200. The lens was cataractous, but the patient found his way around and did some work. At his insistence the cataract was removed, with as little traumatism as possible, after which vision improved. However, a few weeks later vision began to fail and there was a relapse of the inflammation. Glaucoma developed, for the relief of which another operation was performed. The result was total blindness.

Even after the tension has become normal or subnormal and the eye has been quiescent for years, no operation for the improvement of sight should be advised unless there is a definite and urgent indication. E. Fuchs used to tell his pupils that in all his surgical experience there were only two instances of the extraction of cataract from an eye with sympathetic ophthalmia in which he succeeded in getting a permanently good result. He dwelt on the fact that these were operations on eyes free from signs of inflammation over a long period. The reaction after an operation may be delayed for a while, but sooner or later a membrane forms to bridge over any opening made for optical purposes.

TYPES OF EYES IN WHICH SYMPATHETIC OPHTHALMIA DEVELOPS

1. *Eyes with Decreased Tension.*—It is common experience that sympathetic ophthalmia usually develops in soft, atrophic eyes, that is, in those that show a retracted wound, are inflamed and are sensitive to the touch and in which vision is greatly reduced or lost. As decrease in the tension and failing vision come on gradually, the time of enucleation will vary according to the optimism or pessimism of the surgeon.

2. *Eyes with Normal Tension.*—In contradistinction to the type of soft, atrophic eye, there is a type of nonatrophic and nonglaucomatous eye in which after injury the tension remains normal and vision is sufficiently good to warrant retention of the eye. These eyes, too, may give rise to sympathetic ophthalmia and should be kept under observation.

3. *Eyes with Primary Glaucoma.*—There are cases of primary glaucoma in which by operation the tension is reduced to normal, especially by means of a filtering scar, and the organ remains quiescent.

In this group of eyes with primary glaucoma, there are also those in which after an operation inflammation sets in and the eye becomes very soft. This statement refers especially to eyes with absolute glaucoma. One should never congratulate oneself on obtaining very low tension in an eye with primary glaucoma, because abnormal softness of such an eye as a result of inflammation after operation brings it into the danger zone for sympathetic ophthalmia.

4. *Eyes with Secondary Glaucoma.*—There are cases in which after a perforating wound, although the sight has been lost, the eye becomes hard owing to secondary glaucoma. It is not customary to urge enucleation, because eyes with secondary glaucoma are not considered likely to give rise to sympathetic ophthalmia. Nevertheless, sympathetic ophthalmia may develop in these hard eyes, although, as a rule, it sets in a long time after the injury. In the history of such eyes it is often stated that in addition to increase in tension there were attacks of redness and pain for some time, as is recorded in the histories of old, atrophic globes.

HISTOLOGIC SURVEY

The knowledge that sympathetic ophthalmia is not always excited by a soft eye but that occasionally it may be excited by an eye with secondary glaucoma, and the fact that it may be excited by an eye with primary glaucoma, after an operation, led me to suspect from clinical observation that there may be a relationship between a preexisting increase in the tension in the exciting eye and the course and prognosis of the ophthalmia. However, the special incentive for this paper was the impression gained in former histologic surveys that in glaucomatous eyes relatively little of the specific infiltration of sympathetic ophthalmia developed. All told, in material consisting of some one hundred and twelve eyes that had excited sympathetic ophthalmia, thirteen were found in which there had been glaucoma previously. These thirteen eyes fell into the following classifications: eyes with primary glaucoma, which after operation for high tension excited sympathetic ophthalmia (group A) and eyes with secondary glaucoma which after injury or operation excited sympathetic ophthalmia (group B).

GROUP A. EYES WITH PRIMARY GLAUCOMA WHICH AFTER OPERATION FOR HIGH TENSION EXCITED SYMPATHETIC OPHTHALMIA

CASE 1.—*History.*—Four months before enucleation of one eye of a woman 70 years of age iridectomy had been performed. Fourteen days before enucleation of this eye the fellow eye became inflamed. The tension was + 2.

Microscopic Examination.—No typical sympathetic nodules were present in the uvea, although lymphocytic foci were seen.

CASE 2.—*History.*—Two cyclodialyses had been performed a year before enucleation of one eye of a woman, 58 years of age, and a Lagrange operation

and posterior sclerotomy had been performed four months before enucleation. About one month before enucleation of this eye the fellow eye became inflamed. The tension was 21 mg.

Microscopic Examination.—This case proved to be an instance of pure sympathetic infiltration, uncomplicated by the presence of ordinary inflammation. The uvea at the site of one of the cyclodialyses was particularly affected, which seemed to indicate that the virus responsible for the sympathetic ophthalmia had entered there.[3]

CASE 3.—*History.*—Iridectomy had been performed two months before enucleation of one eye of a woman 56 years of age. Fourteen days before enucleation of this eye the fellow eye became inflamed. The tension was normal.

Microscopic Examination.—Typical foci of sympathetic infiltration were seen scattered throughout the uvea and within the walls of the emissaria in the sclera.

CASES 4, 5 and 6.—*Histories.*—On the first two eyes iridectomy had been performed, and on the third an Elliott trephining was done. The tension at the time of enucleation was recorded as normal in the first case. There was no record of it in the other two cases. However, anatomic evidence showed that in all these three cases a previous increase in tension had given way to hypotony. Thus, in the first case the papilla was swollen; in the second case the retina was detached, and in the third case, although an extensive glaucomatous excavation was present, yet, as proof of hypotony, the wound of an iridectomy done six weeks previously was widely gaping.

Microscopic Examination.—The eyes of these three patients showed abundant typical specific infiltration, and in the eye with the gaping wound the infiltration in the iris was so extensive that it was converted into a tumor-shaped mass that clinically might have resembled iris bombé.

In résumé, in five of the six cases of primary glaucoma in eyes that had been operated on, sympathetic ophthalmia developed at a time when the eye had become soft. Histologic examination showed an ample amount of typical specific infiltration in every one of these eyes. In one case, an exception, the disease developed when the eye was still in a state of high tension. This was the sole eye of the entire group that failed to reveal characteristic sympathetic infiltration.

GROUP B. EYES WITH SECONDARY GLAUCOMA WHICH AFTER INJURY OR OPERATION EXCITED SYMPATHETIC OPHTHALMIA

CASE 1.—*History.*—Forty-one years before enucleation of one eye of a man aged 43 years the eye suffered from a perforating ulcer of the cornea, with incarceration of the iris. It had always been blind. It was inflamed a number of times during the intervening years. Two months before enucleation an unusually severe attack of iridocyclitis set in. Five days before enucleation of this eye the fellow eye became severely inflamed. The tension was + 1.

Microscopic Examination.—The iris, including the incarcerated part, showed numerous typical sympathetic nodules, as did the ciliary body and choroid. The other observations were in marked contrast to what one would expect in an

3. Samuels, B.: Significance of Specific Infiltration at the Site of Injury in Sympathetic Ophthalmia, Tr. Am. Ophth. Soc. **30**:253-267, 1932.

eye injured forty-one years before. Within the cavity formed by a detached and folded retina there was a large necrotic abscess in the stage of encapsulation, seemingly not older than two months, which corresponded to the period of recent inflammation recorded in the history. It was believed that about two months before enucleation bacteria pervaded the corneal epithelium and entered the eye along the incarcerated iris, causing the abscess of the vitreous. The fact that in the iris, particularly in the incarcerated part, there were characteristic nodules led to the conclusion that the virus responsible for the sympathetic ophthalmia entered at the same time and through the same channels as the bacteria that produced the abscess. This would be an instance in which in the same eye two different processes, both late in origin, were running their separate courses—the one producing an exudation of pus (the abscess in the vitreous) and the other a specific infiltration (sympathetic ophthalmia).

CASE 2.—*History.*—One eye of a woman 68 years of age was injured eighteen years before enucleation and became blind one year before enucleation. Three months before enucleation sympathetic ophthalmia developed. The tension was + 1.

Microscopic Examination.—Isolated specific nodules were present in the choroid. The nerve head was extensively excavated. There was histologic evidence, in the form of a detachment of the retina, to show that at the time of enucleation the eye was soft.

CASE 3.—*History.*—Seven years before enucleation of one eye of a woman aged 61 years extraction of cataract was performed, and iridectomy was done later because of an incarcerated iris. Six weeks before enucleation sympathetic ophthalmia developed. The tension was + 1.

Microscopic Examination.—Only two small specific nodules were present in the iris. Elsewhere in the uvea there were lymphocytes.

CASE 4.—*History.*—Three years before enucleation the eye was injured by a needle. Later the lens was extracted; this was followed by a prolapse of the iris, which was excised. Four days before enucleation sympathetic ophthalmia developed. The tension was + 2.

Microscopic Examination.—No specific nodules were seen in the uvea; instead only a few lymphocytic foci were present. The optic nerve was deeply excavated.

CASE 5.—*History.*—One eye of a man aged 41 years was injured by a splinter of iron eighteen months before enucleation. Sixteen and a half months before enucleation the eye became hard. Fifteen months before enucleation sympathetic ophthalmia developed. The tension was + 1.

Microscopic Examination.—A few small specific nodules were present in the choroid. The optic nerve showed a glaucomatous excavation.

CASE 6.—*History.*—Seven years before enucleation a piece of wood entered the eye. Six and a half years before enucleation sympathetic ophthalmia developed. Six and a quarter years before enucleation iridectomy and extraction of a cataract were performed. Ectasia of the cornea and sclera developed. The tension was + 1.

Microscopic Examination.—A single typical specific nodule was seen in the iris, and in the choroid there were only pure lymphocytic nodules. The nerve head was pathologically excavated.

CASE 7.—*History.*—One year before enucleation one eye of a man aged 72 years was severely contused, with dislocation of the lens. Because of secondary glaucoma iridectomy was performed. Three weeks before enucleation sympathetic ophthalmia developed.

Microscopic Examination.—Typical foci of sympathetic infiltration were present in the iris. No other part of the uvea showed infiltration. The nerve head was not excavated.

In résumé, in six cases there was surprisingly little specific infiltration in the uvea, and in one case there was none.

As an exception, one eye in this group, which was softened by an abscess of the vitreous and in which it was questionable whether glaucoma had existed for more than a brief period, showed heavy specific infiltration.

DURATION OF THE SYMPATHETIC OPHTHALMIA

The interval between the onset of the sympathetic ophthalmia and the enucleation of the exciting eye was long in three cases, being three months in one, fifteen months in another and six and a half years in a third. Such long intervals may have had a bearing on the amount of specific infiltration seen in the specimens, as the end-result of sympathetic infiltration is the transformation of the areas of the uvea involved into scar tissue. Against the assumption that the infiltration in the enucleated or exciting eye had become regressive is the fact that the uveal stroma was never found to have been converted into scar tissue. This was taken as proof that a more extensive infiltration than that seen had never existed. These cases served to emphasize, above all else, the chronicity of the disease.

TIME OF ONSET OF THE GLAUCOMA

It might be argued that glaucoma was never present at any time before the development of the sympathetic inflammation but that it was a complication of the disease. This theory assuredly is not tenable in case 5 of the group of eyes with secondary glaucoma, because the increase in tension began six weeks after the injury, which was a number of weeks before sympathetic ophthalmia developed. In the other six cases of this group there was anatomic evidence of increased tension, such as ectasia of the sclera and cornea and deep and undermined excavations of the nerve head, proving that in every instance there had been a period of long-lasting glaucoma.

COMMENT

From the survey just given one gleans the following facts:

If after an operation for primary glaucoma inflammation and hypotony set in, the eye has been brought into the realm of eyes potentially capable of giving rise to sympathetic ophthalmia.

My impression that the infiltration is scanty if sympathetic ophthalmia occurs in eyes with primary glaucoma after operation was

not confirmed in the cases studied. There were examples of a large amount of infiltration in the group of eyes with primary glaucoma. However, it is recalled that in five of the six cases of this group the eye was enucleated at a time when hypertonia had given place to hypotonia.

The view that in the case of an eye with secondary glaucoma due to injury or operation there is absolutely no danger as regards sympathetic ophthalmia was not substantiated, for in six cases of the group of eyes with secondary glaucoma sympathetic ophthalmia developed in spite of the increase in tension in the exciting eye at the time. It is understood, however, that the danger that an eye with secondary glaucoma may give rise to sympathetic ophthalmia is less than that one that is soft and atrophic from the start may do so.

My impression that if sympathetic ophthalmia develops in an eye with secondary glaucoma it is characterized by the presence of scanty infiltration in the uvea of the exciting eye was correct. It is probable that the infiltration in the second eye may be correspondingly scanty. This would explain my clinical observation that when the preexisting tension in the exciting eye was high the inflammation in the fellow eye was likely to run a milder course than if it had been excited by an eye of the soft, atrophic type.

Whenever an eye with a history in any way connected with glaucoma is examined histologically after an injury or operation, special search should be made for the specific infiltration, which may be scanty.

EXPERIMENTAL HYPERTENSION

IV. CLINICAL AND PATHOLOGIC STUDIES OF THE EYES: A PRELIMINARY REPORT

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AND
HARRY GOLDBLATT, M.D.
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This communication deals with the pathologic changes in the eyes of dogs with persistent elevation of the blood pressure produced by means of renal ischemia. At the time when the first report [1] was made it was known only that permanent elevation of the systolic pressure resulted from this method. This fact has been confirmed by several publications,[2] but recently it was shown [2d,e] that there is also an elevation of the diastolic pressure in such animals.

This report is restricted to a study of the abnormal changes detected ophthalmoscopically in eight dogs and one monkey with experimental

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From the Institute of Pathology and the Department of Ophthalmology, Western Reserve University.

1. Goldblatt, H.; Lynch, J.; Hanzal, R. F., and Summerville, W. W.: Studies on Experimental Hypertension: I. The Production of Persistent Elevation of Systolic Blood Pressure by Means of Renal Ischemia, J. Exper. Med. **59**: 347 (March) 1934.

2. (*a*) Page, I. H.: Relationship of Extrinsic Renal Nerves to Origin of Experimental Hypertension, Am. J. Physiol. **112**:166 (April) 1935. (*b*) Page, I. H., and Sweet, J. E.: Extirpation of Pituitary Gland on Arterial Blood Pressure of Dogs with Experimental Hypertension, Proc. Soc. Exper. Biol. & Med. **34**:260 (March) 1936. (*c*) Elaut, L.: Hypertension artérielle chronique chez le chien par ischémie rénale, Compt. rend. Soc. de biol. **122**:126, 1936. (*d*) Wood, J. E., Jr., and Cash, J. R.: Experimental Hypertension: Observations on Sustained Elevation of Systolic and Diastolic Blood Pressure in Dogs, J. Clin. Investigation **15**:543 (Sept.) 1936. (*e*) Collins, D. A.: Hypertension from Constriction of the Arteries of Denervated Kidneys, Am. J. Physiol. **116**:616, 1936. (*f*) Harrison, T. R.; Blalock, A., and Mason, M. F.: Effects on Blood Pressure of Injection of Kidney Extracts of Dogs with Renal Hypertension, Proc. Soc. Exper. Biol. & Med. **35**:38 (Oct.) 1936. (*g*) Page, I. H.: Vaso-Pressor Action of Extracts of Plasma of Normal Dogs and Dogs with Experimentally Produced Hypertension, ibid. **35**:112 (Oct.) 1936. (*h*) Prinzmetal, M., and Friedman, B.: Pressor Effects of Kidney Extracts from Patients and Dogs with Hypertension, ibid. **35**:122 (Oct.) 1936. (*i*) Bouckaert, J. J.; Elaut, L., and Heymans, C.: Vasomotor Carotid Sinus Reflexes in Experimental Hypertension Produced by Renal Ischemia, J. Physiol. **89**:3P, 1936.

hypertension for varying periods and to the pathologic changes in the eyes of one of these animals (dog 3-8 of the previous report [1]). This is the only animal so far from which the eyes have been removed during life for a study of the microscopic changes.

COMPLETE REPORT ON DOG 3-8

On April 14, 1932, the main left renal artery of dog 3-8 was constricted. After this there was a moderate rise of the blood pressure. On April 28 the main artery of the right kidney was also moderately constricted. The systolic pressure gradually rose to a much higher level and, with variations, persisted at this level for more than four years (fig. 1). The blood pressure was elevated for two years and ten months before the left eye was excised and for three years before the right eye was removed. Throughout this period there was moderate impairment of renal function, as indicated by the urea clearance, but with variations the nitrogenous products of the blood were usually within normal limits during the period of hypertension.

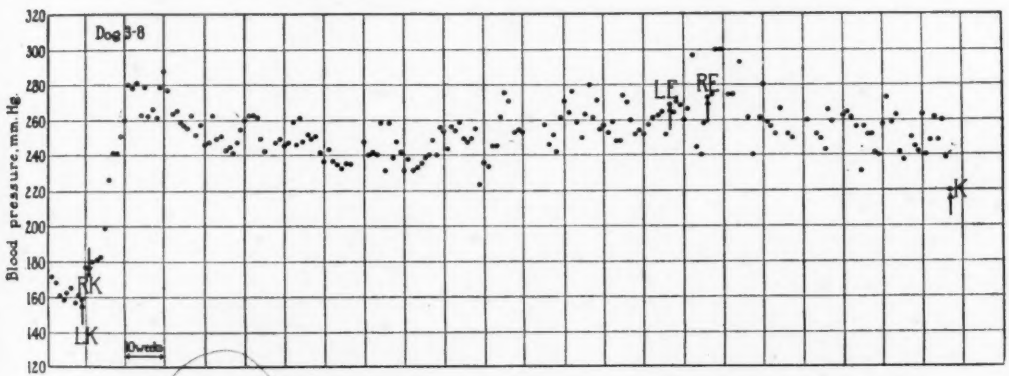

Fig. 1 (dog 3-8).—Chart showing the blood pressure after severe constriction of the left main renal artery (*LK*) and severe constriction of the right main renal artery (*RK*). The left eye was enucleated (*LE*) thirty-four months after the development of hypertension, and the right eye (*RE*) was enucleated thirty-six months after the development of hypertension. The blood pressure remained elevated for fifty months following the constriction of both renal arteries. The dog was killed (*K*) because suppurative metritis with pyometrium developed.

A chronological record of the ophthalmologic observations made on the eyes of dog 3-8 over a period of nearly two years would be voluminous, would involve much repetition and would serve no useful purpose. Briefly summarized, the findings were as follows:

The eyes of dog 3-8 were first examined on April 28, 1933, twelve months after the initial production of hypertension. At that time both eyes were normal. The first pathologic changes in the eyes were noted on Feb. 14, 1934, after the hypertension had existed for one year and ten months. The first pathologic changes were bilateral and consisted of preretinal and superficial retinal hemor-

rhages in the right eye and small areas of retinal edema in the left eye. At this time the retinal arteries appeared smaller than usual in a dog and smaller than when observed on April 28, 1933. Some of the smaller arteries had an unusually keen light reflex and were very wavy. The retinal veins appeared normal. Coincident with the retinal hemorrhage in the right eye, the right optic nerve head became slightly swollen.

The hemorrhage in the right eye was soon followed by more extensive bleeding. Concurrently the edema in the left retina spread rapidly and was associated with a few minute retinal hemorrhages. Four days after the first changes were noted in the left eye, there was a partial detachment of the retina. Three days later the right retina also showed a partial detachment. Both these detachments were located in the lower part of the retina. At this time blood extravasated into the right vitreous. The right nerve head gradually became more swollen, although at no time did the edema of the nerve become sufficient to obscure its edges. The height of the detachment of the retina varied in each eye from time to time. At one period the retina of each eye was almost completely reattached, but it did not long remain so. In the later stages three holes were observed in the detached portion of the right retina. The left retina finally became completely detached. In the left retina, six weeks after the first changes were noted, massive, superficial and deep hemorrhages occurred. In each eye the upper portion of the retina was the site of the most extensive hemorrhage. In this region large hemorrhages occurred on four occasions. While bleeding was noted in the detached portion of the retina, it was not so extensive as in the upper, undetached portion.

The retina of the right eye was free from visible hemorrhage for a few days in January 1935. The retina of the left eye was never entirely clear of blood, although in July 1934 only a few small blood clots were present.

Gradually the blood vessels of the nasal retina of each eye were partly or completely concealed, excepting close to their origin at the nerve head, by a slowly developing white haze, which later became more dense and sheathlike. In the left eye the first pathologic change noted was along the vessels of the nasal retina and was the forerunner of the condition just described. In the right eye small hemorrhages along the vessels of the nasal retina left white streaks or patches when they became depigmented, and these were succeeded by the gradual development of a white sheath or membrane around the vessels. In each eye the development of this white coating of the mesially situated blood vessels was associated with recurrent small hemorrhages which seemed in most instances to leave white or slightly pigmented scars.

The right optic nerve early became moderately swollen and remained so for about nine months. At no time, however, were the retinal vessels on the nerve head entirely obscured, nor did the swelling visibly spread beyond the borders of the optic disk.

The vitreous began to show chocolate-colored blood "floaters" in the right eye sixteen days after the first retinal hemorrhage was observed, while that of the left eye did not show an appreciable number of floaters until after the third large retinal hemorrhage, which occurred six months after the first. It was possible to observe closely a hemorrhage of the vitreous which was attached to the retina in the right eye. It lost its chocolate color within one month, but a characteristic grayish-black floater was still attached to the retina. The vitreous in each eye finally became so full of blood and hemorrhagic débris that the

details of the fundus became indistinct, and the intra-ocular tension began to rise. This complication occurred in both eyes after the fourth series of large hemorrhages in the upper retinal region. In the right eye this stage was reached about eleven months after the first intra-ocular pathologic change had been noted. In the left eye about eight months elapsed before the tension became elevated. Following these occurrences in the left eye, blood appeared in the aqueous, and the tension to touch gradually increased. The eye also became tender to the touch and perhaps painful. The posterior surface of the cornea became blood stained. Early in January 1935 massive intra-ocular hemorrhages occurred, and acute secondary glaucoma supervened. This was associated with protrusion of the eyeball and ulceration of the cornea. Five days later, on January 7, two years and ten months after the production of hypertension, the left eye was enucleated to prevent perforation of the corneal ulcer.

In the right eye, after large retinal hemorrhages on January 16, the vitreous and aqueous gradually became blood stained, and the details of the fundus were almost indiscernible. By March 15, exactly three years after the production of hypertension, as the intra-ocular tension in the right eye was becoming elevated it was decided to remove the eye to avoid the destruction that would inevitably have followed more hemorrhages or the establishment of secondary glaucoma.

Gross Examination of the Eyes.—Right Eye: The eyeball was soft and indented readily. The retina was detached and lay on a semisolid vitreous.

Left Eye: The eyeball was firm, and the cornea bulged. The center of the cornea was occupied by a deep ulcer with sloping edges and a shaggy base. The anterior portion of the eye was hyperemic. The vitreous was chocolate colored. The retina was detached.

Microscopic Examination of the Eyes.—Technic: Serial sections of both eyes were cut. Every fifth section was stained. In important portions of the eye all the sections were stained. The following stains were used: hematoxylin and eosin, Verhoeff's stain for elastic tissue, Mallory's stain for connective tissue and Perl's prussian blue stain for hemosiderin. The right eye was the more valuable specimen. The left eye was too disintegrated to permit detailed study.

Right Eye: The cornea and sclera were essentially normal. The chief changes in the iris were in the blood vessels. In some arterial blood vessels the media was definitely thicker than normal. Occasional diseased arteries were present in the ciliary body and at the root of the iris. The choroid was denser than normal and was deeply pigmented. Anteriorly, near the ciliary body, there were areas of moderate edema. There were also other areas where the otherwise detached retina was adherent to the choroid. Some of the choroidal arterial blood vessels showed a definite increase in thickness of their walls, which was apparently confined to the media. In some instances the walls of the vessels show some hyalinization.

The retina was completely detached except in the region of the posterior scleral (optic) foramen, the ora serrata and certain chorioretinal scars. The pigment layer of the retina was attached to the choroid. As a whole, the retina was atrophic. The rod and cone layer had disappeared. A few degenerated ganglion cells remained. Müller's supporting fibers and the nuclei of the internal

and external nuclear layers were clearly visible. The nerve fiber layer contained pigment-bearing cells. These cells and free granules of pigment were also present around deep, as well as superficial, retinal vessels. Certain portions of the retina were cystic, especially in isolated areas near retinal scars and at the ora serrata. All the layers of the retina, except the nerve fiber layer, were involved in this cystic process.

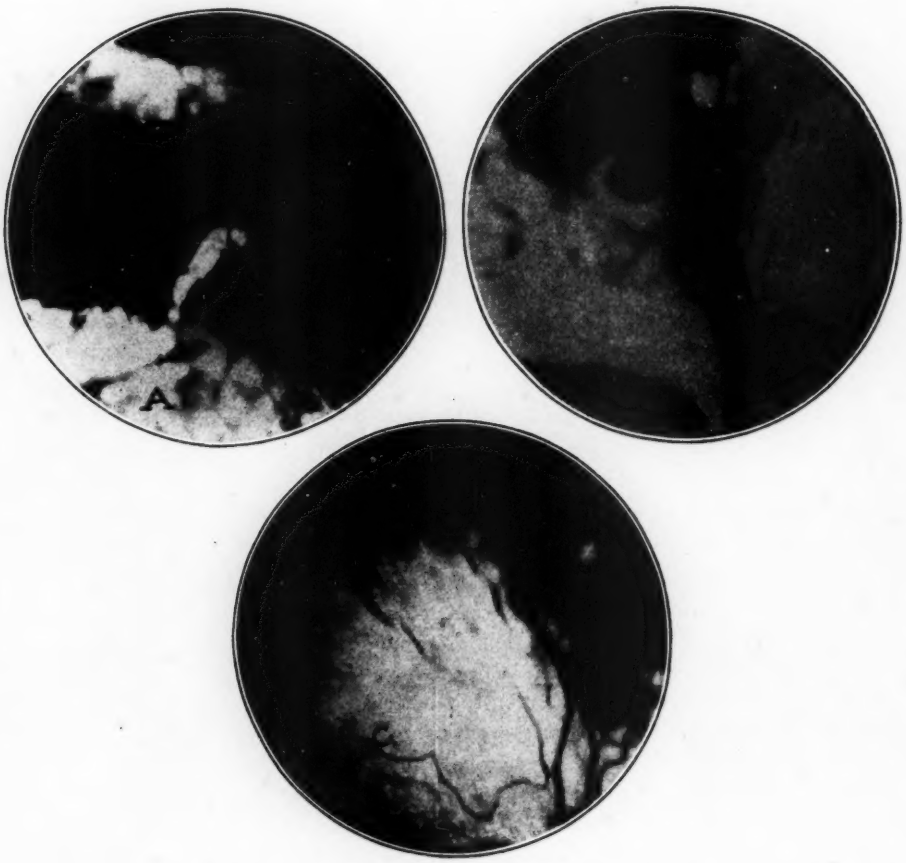

Fig. 2 (dog 3-8).—Photographs of the fundus of the right eye taken on Feb. 16, 1934. *A* shows the optic nerve and the ascending retinal blood vessels. There is a large parapapillary hemorrhage that extends over to, but is not in contact with, the nerve head. Near the upper periphery of the photograph is a second large hemorrhage, which covers the ascending retinal blood vessels. B is a view taken nearer the periphery, along the ascending retinal blood vessels. Attention is directed to a circumscribed hemorrhage surrounded by a white zone. In this region a closed arteriole was observed with the ophthalmoscope. The blood vessels shown in figures 3, 4, 5, 6, 7 and 8 occurred in this area. Recurrent hemorrhages were observed along the ascending retinal blood vessels in the region shown in this photograph. *C* is a view taken slightly higher in the fundus. It shows the ascending retinal blood vessels partly covered by hemorrhage and edema of the retina.

There were several retinal scars, some of which were attached to the choroid. One of these scars was associated with a local detachment of the choroid. In the posterior portion of the eye serial sections passed through a retinal scar, which was studied clinically. In this region deep and superficial retinal hemorrhages occurred, and a small, sclerotic, apparently occluded blood vessel was seen with the ophthalmoscope. The retinal scars varied considerably in density, the degree of variation depending apparently on the portion of the retina most invloved. Where the nerve fiber had multiplied at the expense of the external layers the scar consisted of a rather open network of fibroblasts and neuroglia cells containing diseased blood vessels, Müller's fibers, blood pigment and pigment-bearing cells. Where the proliferation was confined more to the outer layers of the retina the scar was more dense, consisted of masses of nuclei of the cells of the external and internal nuclear layers and was otherwise like the

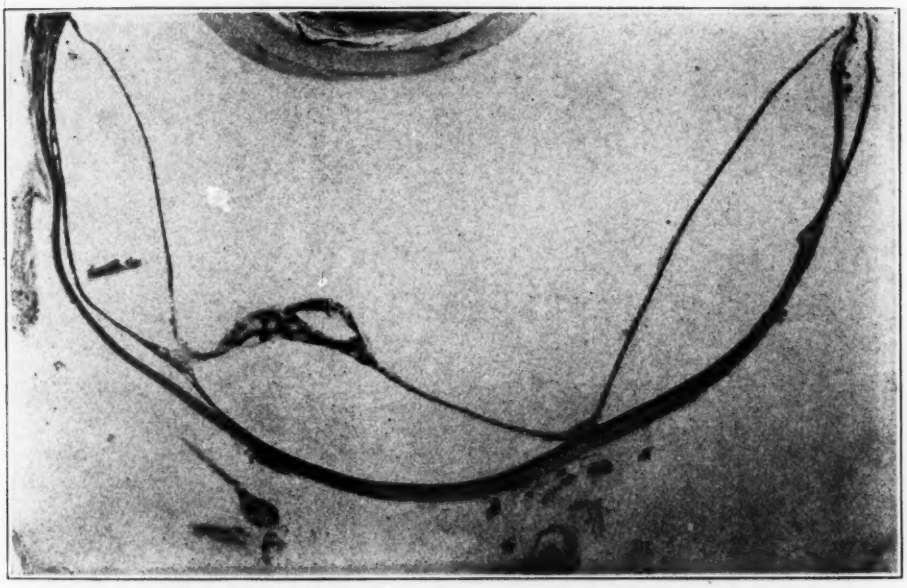

Fig. 3 (section 165).—Section of the posterior portion of the right eye immediately above the optic nerve head. A large scar is located on the nasal side of the eye in the degenerated detached retina. It contains two diseased blood vessels with hyalinized walls. The periphery of the scar is adherent to the slightly edematous locally detached choroid.

scar just described. Some of the arteries and the tissue surrounding them appeared to be hyalinized.

The changes in the retinal vascular system involved all sizes of arterial blood vessels. The main retinal arteries on and near the optic nerve head and their terminal branches at the ora serrata, and arterioles in retinal scars, showed the most striking vascular disease. The intima was principally affected. It was greatly thickened. In some vessels the intima narrowed the lumen almost to occlusion; in others the lumen appeared to be closed. The thickening of the media in some vessels was due to varying degrees of hypertrophy and hyper-

plasia of the muscle, which was the seat of varying degrees of hyalinization. While some thickening of the intima was general, the degree of thickening and degeneration was not uniform, even in the same blood vessel, which corroborates the observation of Moritz and Oldt [3] in cases of arteriolar sclerosis in man. Blood vessels were present in which the lumen was closed, but on the proximal

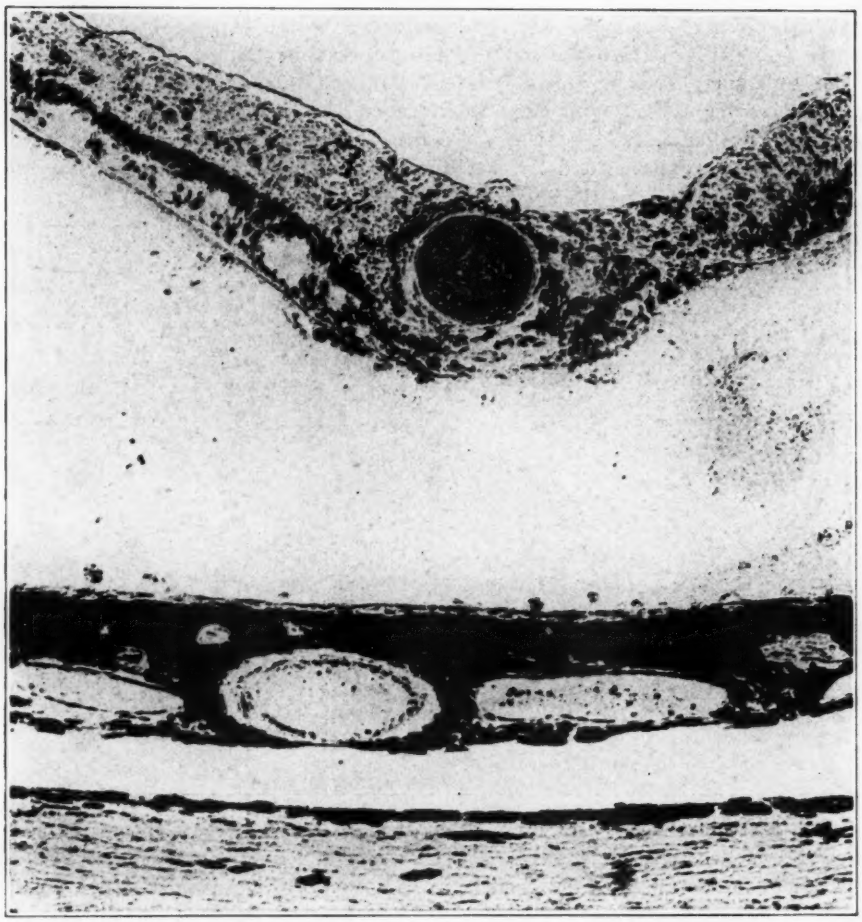

Fig. 4 (section 125).—Section of the right eye, showing an arterial blood vessel located in the lower portion of a posterior retinal scar. In this region of the retina recurrent deep and superficial hemorrhages occurred. An occluded arteriole was also observed ophthalmoscopically. The lumen of this vessel is represented by the triangular shaped clump of cells of the intima at its center. The entire wall is hyalinized and stained pink with eosin. Surrounding the blood vessel is some brown and golden pigment, much of which took the stain for hemosiderin. × 165.

3. Moritz, Alan R., and Oldt, Mary R.: Arteriolar Changes in Essential Hypertension, Am. J. Path. **11**:885 (Sept.) 1935.

and distal side of the occluded region the vessel was patent, although the wall was abnormally thick. No duplication of the vascular elastic lamina was observed in any of the vessels.

Many isolated, small hemorrhages associated with small clumps of golden brown pigment were present in the retina. Extravasated blood was present in both the superficial and the deep layer of the retina. In some instances the hemorrhage extended outward to the external limiting membrane. There was considerable iron-free and iron-containing perivascular pigment, which was mostly

Fig. 5 (section 109).—Section of the right eye, stained to demonstrate the presence of hemosiderin, showing portions of the retina, choroid and sclera. The black dots and areas in the retina are hemosiderin. The pigment in the choroid and sclera is not iron-containing pigment. The retinal blood vessel with a thick, partly hyalinized intima is a distal section of the same blood vessel shown in the ciliary body was detached from the sclera and folded backward. On the

extracellular. In the lower portion of the retina blood vessels of capillary size were outlined by fine granules of dark iron-free pigment which impregnated their walls.

The subretinal space contained an amorphous, structureless material and partly absorbed blood. In this material were a few scattered lymphocytes and round cells containing blood pigment.

In the anterior portion of the vitreous several small hemorrhages were observed. With Mallory's elastic tissue stain the vitreous was shown to be full of structureless strands. Some of these strands were attached to the ciliary body anteriorly and to a retinal scar posteriorly.

The anterior chamber was empty; the filtration angle was normal.

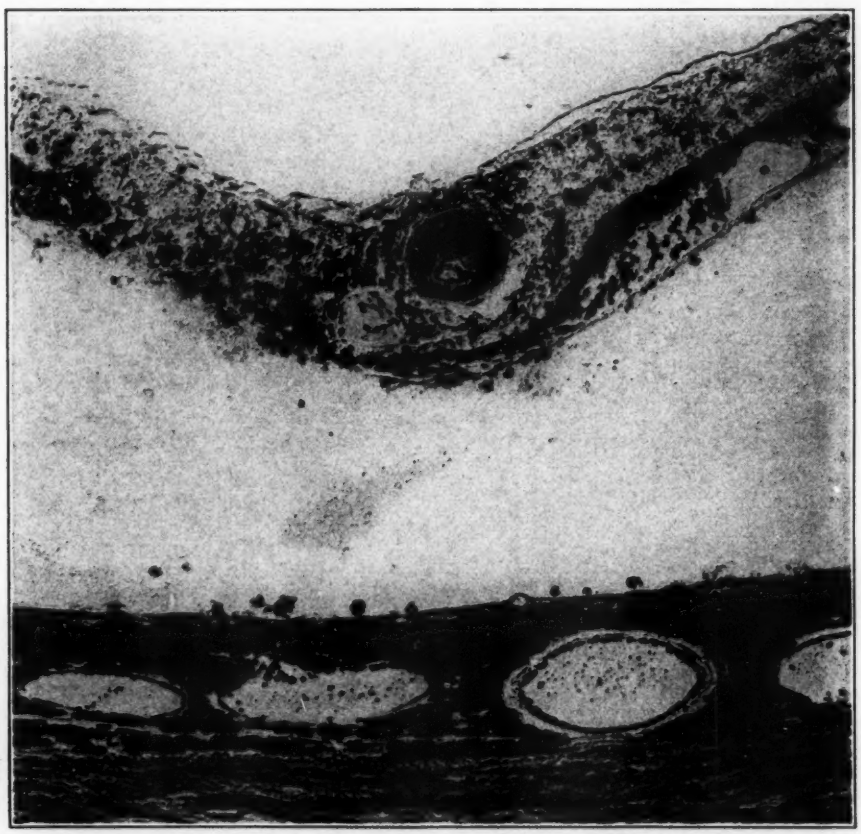

Fig. 6 (section 131).—Section of the right eye slightly nearer the nerve head, showing the same retinal arterial blood vessels shown in figure 4. In this part the vessel has a small but definite lumen. × 165.

There was a small zone of fragmented fibers in the posterior part of the lens.

The intra-ocular portion of the optic nerve projected slightly into the cavity of the eye. The portions of the retinal artery and its branches seen in the optic nerve exhibited moderate thickening of the media. The posterior ciliary arteries also showed a definite increase in the diameter of the tunica media, but the degree of change was not nearly as great as in some of the retinal vessels.

The reaction for iron was given by various-sized particles in the posteriorly located retinal scar and by similar particles (hemosiderin) scattered unevenly throughout the inner layers of the retina. The isolated retinal scars contained little iron-containing pigment. A chorioretinal scar on the nasal side of the eye contained less hemosiderin than did the posterior retinal scar. Except for a little pigment in the head of the optic nerve, the other structures of the eye were practically free from hemosiderin.

The diagnosis of the condition of the right eye was: arteriolar sclerosis, especially retinal; chronic retinitis; detachment of the retina; partial detachment of the choroid, and incipient cataract.

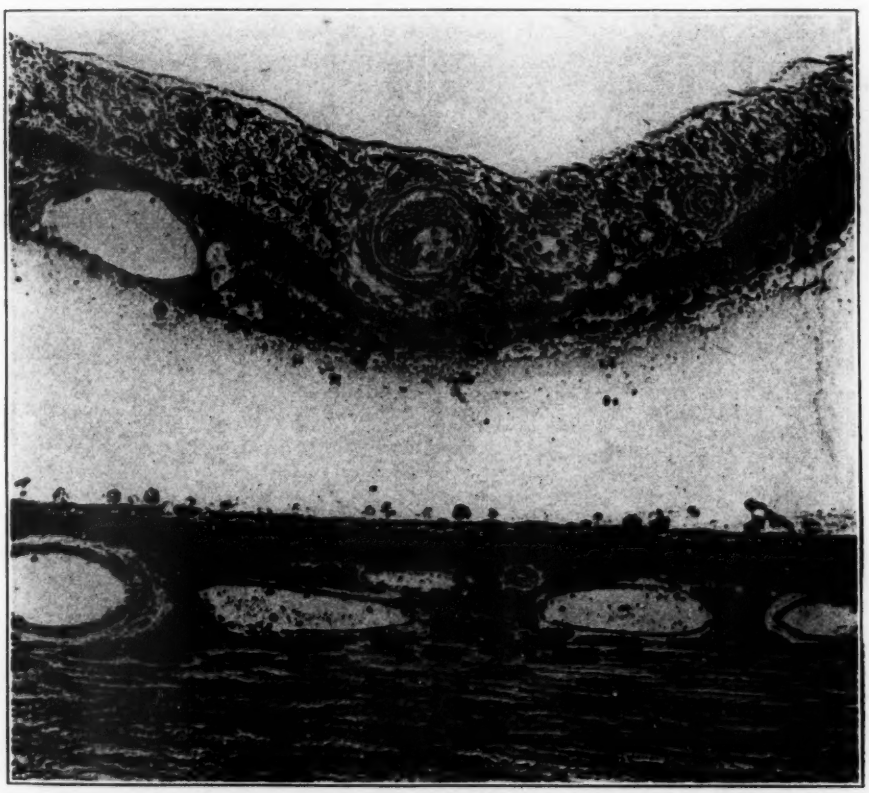

Fig. 7 (section 150).—Section of the right eye, showing the proximal portion of the same retinal blood vessel shown in figure 4. The lumen is narrowed by the encroaching intima, but it is even larger than that shown in figure 6. × 165.

Left Eye: Because the left eye was not removed early, its value as a pathologic specimen was greatly reduced. The histologic picture of the left eye was that of great destruction.

The central portion of the cornea was occupied by a large ulcer on the verge of perforation. Keratectasia was so extensive as to resemble keratoglobus.

At its deepest point the ulceration was so deep that the base was separated from the anterior chamber by only a few strands of posterior stromal connective

tissue, over a portion of which Descemet's membrane was absent. Descemet's membrane had ruptured behind the ulcerated portion of the cornea. A peripheral portion of this membrane had separated from the cornea. It lay partly coiled in the deep anterior chamber. The proximal end was attached by a strand of connective tissue to the root of the iris. The base of the corneal ulcer was occupied by necrotic tissue, exudate and granulation tissue. The edges of the ulcer showed massive infiltration by polymorphonuclear leukocytes and many small newly formed capillaries.

The sclera was slightly thinner than normal.

The iris presented a reverse iris bombé, due apparently to distention of the anterior chamber of the eye by blood. On the side of the eye where Descemet's

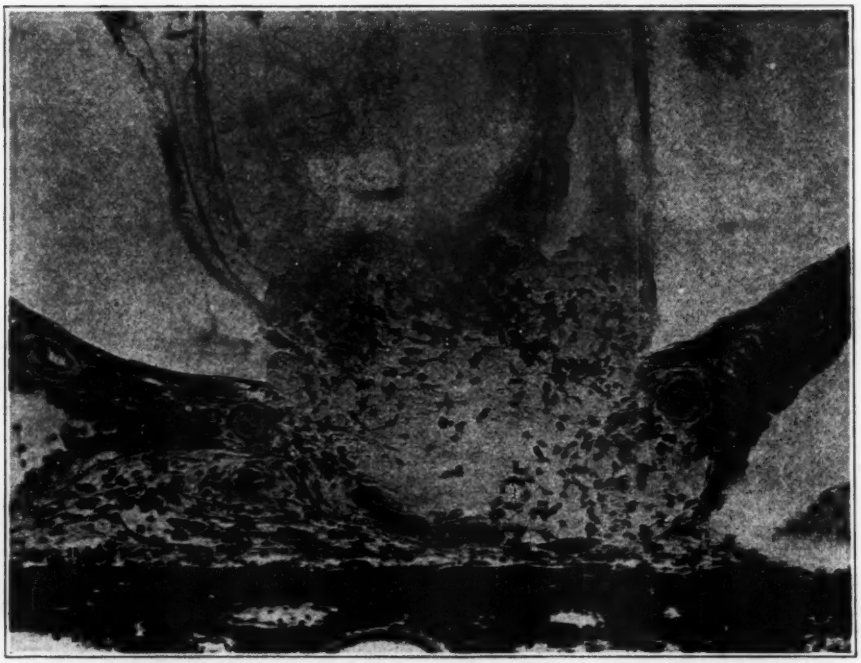

Fig. 8 (section 90).—Section of the right eye, showing posterior retinal scar situated above the nerve head. At both edges of the scar are occluded or nearly occluded blood vessels. Strands of tissue extend from the scar into the vitreous cavity. A large quantity of hemosiderin is present. This section is from the portion of retina shown in figure 1 *B* and *C*. × 135.

membrane was detached from the cornea the root of the iris and the anterior portion of the ciliary body were detached and pressed backward against the crystalline lens. The iris was atrophic and edematous. The blood vessels of the iris were distended by blood. The ciliary body had degenerated. It was edematous and hyperemic. On one side of the eye the anterior two thirds of the ciliary body was detached from the sclera and folded backward. On the opposite side the ciliary body was distended by a blood clot.

The posterior two thirds of the choroid was detached on the same side of the eye as the detached ciliary body. The general appearance of the choroid was that of atrophy and degeneration, with associated edema, hyperemia and extravasation of blood in the anterior portion.

The retina was detached except at the ora serrata and the optic nerve. It was so severely degenerated that only the vaguest structural details remained.

The optic nerve showed atrophy in certain sections. There were areas of hemorrhage beneath the nerve sheath. The proximal portion of the nerve contained scattered iron-free pigment and iron-containing pigment.

The crystalline lens occupied a position nearly corresponding to the center of the eye. It was apparently pressed and held backward by the hemorrhage in the anterior chamber and the posteriorly bulging iris. There were a few small areas of fragmented subcapsular lenticular fibers at the anterior and the posterior pole of the lens.

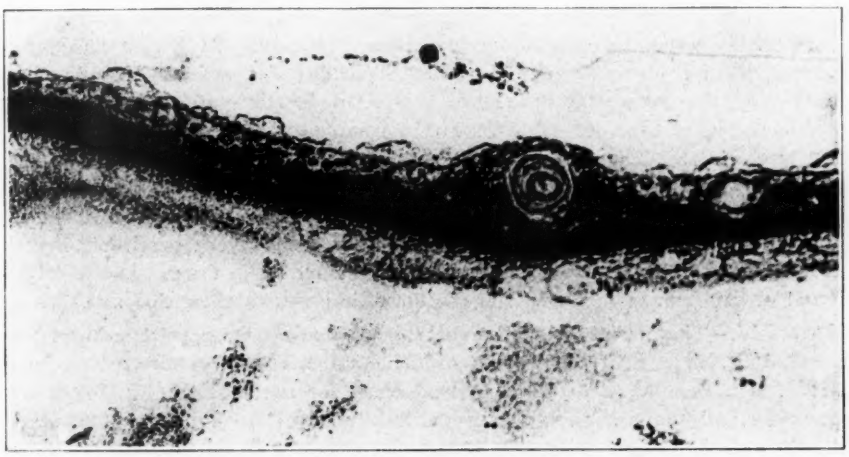

Fig. 9 (section 220).—Section of the right eye, showing small portion of the retina near the ora serrata. Located in the inner layers is a small arteriole, partly surrounded by clumps of iron-containing pigment. The lumen of the blood vessel is very small. The vessel wall is very thick. Subretinal exudate is present. × 158.

The anterior chamber, the space between the inner surfaces of the detached retina, the subretinal space and the subchoroidal space were filled by blood clots of various ages.

The blood vessels in this eye presented little of interest, as those of the degenerated retina were hardly identifiable. Their outline could frequently be discerned, however, owing to a deposit of fine brown pigment in their walls. The ciliary arteries showed an increase in the thickness of the media. This thickening of the media was also discernible in some of the large choroidal arteries.

The diagnosis of the condition of the left eye was: ulceration of the cornea, keratectasia, partial detachment of the choroid and ciliary body, detachment and degeneration of the retina, massive intra-ocular hemorrhage and atrophy of the optic nerve.

SUMMARY OF OPHTHALMOLOGIC FINDINGS IN SEVEN OTHER DOGS AND ONE MONKEY

Dog 6.—The normal state of this dog's eyes was not determined. The final operation for constriction of the renal arteries was performed on Jan. 16, 1931. Since then the animal has had a moderate degree of persistent hypertension, the systolic pressure being usually in the neighborhood of 220 mm. of mercury. This animal's eyes were first examined on Nov. 8, 1935, at which time periarteritis was present. The arteries were tortuous. There was an increase in the intensity and width of the arterial light reflex. The vitreous was a little hazy, owing to synchysis scintillans. Subsequently, the arterioles became irregular in outline. A few small arterial vessels appeared occluded. There have been a few retinal hemorrhages. No edema of the retina proper had occurred. The ophthalmoscopic picture presented so far is one of gradually increasing arteritis or periarteritis, combined with degeneration of the vitreous.

Dog 5-9.—The final constriction of the renal arteries was accomplished on Oct. 18, 1932. On March 2, 1934, the first ophthalmoscopic examination was made. The arterioles were then suggestively tortuous. By July 18 the tortuosity of the small arteries was more obvious. On September 7 there was definite perivasculitis of the peripheral arterioles, associated locally with a small retinal hemorrhage and a zone of edema. The perivasculitis has slowly increased in extent and intensity. Small retinal hemorrhages have occurred frequently, particularly along the diseased blood vessels. Retinal hemorrhages along the blood vessels associated with perivasculitis appear to intensify the vascular covering. Synchysis scintillans is present. This animal has had severe persistent hypertension for more than four years, the systolic pressure being usually in the neighborhood of 270 mm. of mercury, and the diastolic pressure about 200 mm.

Dog 1-17.—The normal condition of this animal's eyes was determined on Dec. 20, 1933. The final constriction of the renal arteries was effected on June 11, 1934. On Feb. 12, 1936, there was observed for the first time an intensified light reflex and slight irregularity of outline (caliber), together with moderate tortuosity, of the retinal arteries. At present this dog is in an early stage of retinal arterial vascular disease. This animal has moderate hypertension.

Dog 1-34.—This animal's eyes were first examined on April 25, 1934, fourteen days after the final constriction of the renal arteries. At that time the eyes were normal. On Feb. 19, 1935, after a period of increasing tortuosity of the small retinal arteries, an arteriole of irregular caliber appeared greatly constricted and possibly closed at one point. At present there is a suggestion of a perivascular sheath. This animal has moderate hypertension.

Dog 2-31.—This dog's eyes were first examined on Nov. 27, 1935, and were normal. The second renal artery was constricted on Jan. 7, 1936. By May 8 there was a definite increase in tortuosity of some of the small retinal arterial blood vessels. Two small retinal hemorrhages have occurred. Perivascular sheaths are developing. This animal has moderate hypertension.

Dog 2-35.—The first examination was on Nov. 27, 1935; the eyes were normal. The second renal artery was constricted on March 11, 1936. On March 17 a small temporal artery with a definite perivascular gray-white sheath was observed Since then several arterioles have become irregular in outline and are showing perivascular coatings. This animal has had continuous severe hypertension, the systolic pressure being usually around 250 mm. of mercury.

Dog 2-50.—This dog's eyes were first examined on May 28, 1936, and were normal. The second renal artery was constricted on September 3. On September 16 the arterioles of the nasal portion of the retina appeared a little tortuous and somewhat indistinct. The blood pressure rose quickly to high levels. On October 10 the systolic pressure as determined by the van Leersum method was 300 mm. of mercury, while the mean pressure as determined by the direct method was 265 mm. of mercury. On October 12 the systolic pressure was more than 300 mm. and the mean pressure more than 280 mm. On October 15 the systolic pressure was above 300 mm. On this day bilateral intra-ocular hemorrhage was found, and the animal was evidently blind. On October 16 the animal was in collapse, and some bloody fluid was coming from the anus, but even in this state the mean pressure was still 240 mm. The animal was killed on this day. The eyes are being sectioned.

Monkey 5.—The final operation to produce renal ischemia was performed on Sept. 25, 1935. This animal's eyes were first examined on May 8, 1936. At this time no abnormalities were noticed, except that the light reflex on the arteries was rather keen. On June 5 bilateral localized areas of edema and scattered retinal hemorrhages were observed. After this the arterial light reflex became wider and more pronounced. The retinal hemorrhages and edema gradually disappeared. Concurrently the retinal arterial system exhibited rapidly increasing evidence of disease. Some retinal arteries assumed a copper wire appearance. Several small arteries resembled silver wires or threads and had areas of occlusion. The blood vessels in this animal's eyes did not become tortuous and were not markedly irregular in outline. This animal died of acute enteritis on December 9. The eyes are being sectioned. The animal had severe hypertension from the time the second renal artery was constricted. The systolic pressure was usually above 300 mm. of mercury, and the diastolic was about 220 mm. during the last six months of the life of this animal.

SUMMARY

Changes visible with the aid of the ophthalmoscope appeared in the eyes of eight dogs and one monkey after varying periods of experimental hypertension due to renal ischemia. The retinal arterial vascular system was apparently primarily affected, with or without the appearance of hemorrhages and edema in the retina.

In one animal (dog 3-8), the eyes of which were examined microscopically, hypertension had existed for three years before the eyes were enucleated. In this animal the pathologic alterations in the retinal blood vessels were noted in the smallest, as well as in the largest, branches of the arteries and in the distal, as well as in the proximal, branches. The principal change in the arterial blood vessels of the retina occurred in the intima. The disease process varied in intensity in the same blood vessel. A blood vessel that was completely closed at one point had a lumen of fair size elsewhere in its course. More diseased blood vessels were noted in the retina than in the choroid, iris or ciliary body. The disease process in the small arteries of the retina varied from simple

hypertrophy and hyperplasia of the muscle to advanced hyalinization of the intima and atrophy of the media.

Hemorrhages which occurred in the deeper or external portions of the retina were absorbed more slowly than those in the superficial or internal portion of the retina. Moreover, the latter, except for floaters in the vitreous, did not seem to leave many traces, while the former, in some instances at least, were succeeded by white zones or zones of iron-free pigment and iron-containing pigment. The small dense hemorrhages along the retinal blood vessels in the horizontal nasal meridian became white, while similar hemorrhages along the vertical blood vessels left pigmented remains. In this respect it is to be noted that the circumscribed hemorrhages in the upper part of the retina were situated on the tapetum. Small, deeply situated, round or oval retinal hemorrhages were associated with arterioles that showed disease visible with the ophthalmoscope. These hemorrhages required a longer period for depigmentation than did the superficial retinal hemorrhages. In the resultant retinal scars there was a considerable amount of iron-containing pigment.

Hemorrhages into the vitreous bleached rather slowly and in many instances left floaters. As the vitreous became loaded with foreign material the tension of the eye tended to increase.

When a white haze appeared on a blood vessel, it never disappeared but gradually became more dense until it concealed portions of the blood vessel. These vascular coverings first became visible toward the distal end of the blood vessel but never reached the proximal end at the nerve head.

The detachment of the retinas was apparently due to retinal and subretinal edema.

OCULAR MANIFESTATIONS OF MALIGNANT NASOPHARYNGEAL TUMORS

REPORT OF CASES

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NEW YORK

Considerable has been written about nasopharyngeal tumors in journals on rhinology, otology, neurology, general surgery and pathology, but there is practically nothing on this subject in the ophthalmic literature. In many cases the first symptoms are referred to or about the eyes. The ophthalmologist may be the first to be consulted in cases of a nasopharyngeal growth, or he may be called on to explain the ocular signs. This form of tumor is not uncommon, and it is important for the ophthalmologist to acquaint himself with the condition. In some cases the group of symptoms immediately suggest the diagnosis, but often there may be only one, two or three symptoms which should make one think of the possibility. The diagnosis is usually difficult, but if the subject is borne in mind and a careful examination is made, the condition will be diagnosed more frequently.

New,[1] in his discussion of the subject, emphasized three important facts: 1. Malignant tumor of the nasopharynx is much more common than is believed. 2. The syndrome which it presents is not generally known, which accounts for the fact that many patients are treated medically and surgically without discovery of the tumor. 3. There is a striking lack of nasal or nasopharyngeal symptoms in many of the cases.

New reviewed seventy-nine cases of nasopharyngeal tumor in patients from 4 to 66 years of age. Twenty-one of the patients had symptoms referable to the eyes; twenty-nine complained of the ears; thirty-eight presented nasal or nasopharyngeal symptoms, and fifty-one had enlarged glands of the neck. In the patients who showed ocular symptoms the following lesions were found: most commonly, diplopia, particularly paralysis of the sixth nerve; ptosis; sometimes complete ophthalmoplegia, with choked disk; atrophy of the optic nerve, and paralysis of a portion of the sympathetic nervous system (Horner's syndrome).

Often a tumor in the nasopharynx is so small that it cannot be detected, but nevertheless there may be extensive involvement of cranial

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Read before the Section of Ophthalmology of the New York Academy of Medicine, Feb. 15, 1937.

1. New, G. B.: Syndrome of Malignant Tumors of the Nasopharynx: A Report of Seventy-Nine Cases, J. A. M. A. **79**:10-14 (July 1) 1922.

nerves and metastasis to the cervical region. The literature is so full of confusion and contradictions in the designation of these growths that it is often difficult to understand what is meant by the terms that various authors have employed. Salinger and Pearlman [2] examined most of the reports available on this subject and found that nasopharyngeal tumors roughly fall within three groups: carcinoma, sarcoma and endothelioma. The subdivisions of these groups are numerous and add much to the confusion of the terminology.

The tumor is generally located in the fossa of Rosenmüller. Its chief characteristic is to spread widely beneath the mucous membrane, so that involvement of the mucous membrane in the nasopharynx, with ulceration and hemorrhage, frequently occurs only late in the disease. The growth burrows into the deeper planes and spreads in all directions. It involves the eustachian tube, the vault of the pharynx, the sinuses, the sphenomaxillary and pterygopalatine fossae, the sphenoid fissure, the orbit and the structures at the base of the skull. Lymphatic chains in the upper pharyngeal and cervical regions are also involved.

The symptoms of nasopharyngeal tumor are produced by involvement of the structures just mentioned, so that the symptoms may be varied.[3] They may be divided into nasal and extranasal. Nasal symptoms may be manifested as bulging of the soft palate, nasal obstruction, dysphagia, hemorrhage, foul odor and taste, alterations of the nasal voice, obstruction of the eustachian tube, tinnitus and obstructive deafness. Extranasal symptoms are commonly present in the early stages of the disease. The usual signs and symptoms are: pain in the eye, forehead, side of the face and temporal region; toothache; earache; tinnitus; a sense of fulness in the ear; diplopia; blindness; proptosis; paresthesia of the face; enlarged cervical glands; dysphagia; aphonia; hoarseness, and metastasis to distant parts.

The nasopharynx should be examined in all patients with any of the aforementioned symptoms. A tumefaction that brings the side on which a tumor is suspected to be present into greater prominence than the opposite side should warrant a biopsy, because this growth is located beneath the epithelial surface.

The prognosis is poor. The symptoms are alleviated by roentgen treatment, but eventually the condition is fatal in all cases.

In regard to treatment, the tumor cannot be reached surgically. Radium and roentgen therapy are used. J. Maimin and H. Vassiliadis [4] stated the belief that internal treatment is a valuable adjunct in the

2. Salinger, S., and Pearlman, S. J.: Tr. Am. Acad. Ophth. **40**:281-316, 1935.

3. Hansel, F. K.: Malignant Tumors of the Nasopharynx, Arch. Otolaryng. **9**:12-22 (Jan.) 1929.

4. Maimin, J., and Vassiliadis, H.: Strahlentherapie **54**:193-214 (Oct. 16) 1935.

radiotherapy of tumors. They have been able to demonstrate the beneficial influence of the internal administration of barium on tar cancers in mice. In the clinic for patients with tumor at Löwen, the roentgen treatment is based on the principles of Coutard. Teleradium and other forms of radium treatment are used, and barium saccharin is given by mouth.

In this article a review of thirty-eight cases is given, in five of which the patient first consulted the ophthalmologist. These cases were observed at the Mount Sinai Hospital. Twenty-two of the patients had only local signs—signs in the nasopharynx and ear—and metastasis to the cervical region. In this group thirteen had enlarged cervical glands. The only ocular sign was unequal pupils in three cases. Sixteen patients had symptoms referable to the eye; this group is of particular interest. The ocular signs were as follows: involvement of the fifth nerve (one or more branches), in twelve cases; Horner's syndrome (complete or incomplete), in ten cases; involvement of the sixth nerve, in eight (in one the condition was bilateral); involvement of the facial nerve, in five; ophthalmoplegia interna and externa, in four (in two the condition was unilateral and in two bilateral); exophthalmos, in three (in two the condition was unilateral and in one bilateral); ptosis, in two; papilledema, in one; papillitis, in one; atrophy of the optic nerve, in one; a mass in the orbit, in one. Two of the patients had the syndrome of the sphenoid fissure, i. e., paralysis of the third, fourth and sixth nerves and of the ophthalmic division of the fifth nerve.

Other signs in this group were referable to the ear in fourteen cases; the nasopharynx was affected in six, and there were swelling in the neck in five, deviation of the tongue in three, impairment of taste in four and impairment of smell in four. There also occurred neuralgia of the sphenopalatine nerve, dysphagia, dysarthria, atrophy of one side of the tongue, paralysis of one side of the pharynx, hoarseness, paralysis of the masseter muscle, paralysis of the pterygoid muscle, paralysis of the glossopharyngeal and the vagus nerve and involvement of the sternocleidomastoid muscle. Two patients had syndrome of the jugular foramen, paralysis of the ninth, tenth, eleventh, and twelfth nerves.

The nerves most commonly affected were the fifth nerve, a portion of the sympathetic nervous system, the sixth nerve and the facial nerve.

The diagnoses considered in some of these cases were as follows: mastoiditis, retrobulbar neuritis, sinusitis, neuralgia of the sphenopalatine nerve, syphilis, a cerebral neoplasm, an expanding tumor, encephalitis, an infectious process involving the meninges, an intrasellar or suprasellar growth, and chronic thrombosis of the cavernous sinus.

In this group the roentgen rays were helpful in the diagnosis in eight cases. In several cases there was destruction of bone. In some cases the diagnosis of tumor was confirmed by the roentgenologist.

The following operations were performed before the diagnosis was made: operation on the nasal septum and on the nasal accessory sinuses, paracentesis of the ear, extraction of teeth, mastoidectomy, injection into the sphenopalatine ganglion, lumbar puncture and craniotomy. In some of the cases several operations were done.

The types of tumor were practically the same in the two groups of cases. For the patients with local signs the reports of pathologic examination were: carcinoma of the nasopharynx, immature squamous cell carcinoma, squamous cell carcinoma of the ethmoid and sphenoid sinuses, sarcoma (large round cell) of the nasopharynx, transitional cell carcinoma and lympho-epithelial carcinoma. For the patients with ocular signs the reports of pathologic examination were: carcinoma of the nasopharynx, unripe squamous cell carcinoma, squamous cell carcinoma, transitional cell carcinoma, lympho-epithelioma, embryonal cell carcinoma and lymphosarcoma. In some cases several biopsies were necessary before the tumor was observed.

COMMENT

All the cranial nerves affected were involved at the foramina at the base of the skull.

The sympathetic nervous system may be involved in the neck, owing to metastasis to the cervical region. However, among the sixteen patients with ocular signs, the sympathetic nervous system was involved in ten, and the cervical glands were enlarged in only five. In the group of patients with local lesions, thirteen had glandular involvement, and only three had unequal pupils. Undoubtedly in many cases the sympathetic nervous system is involved higher up than the cervical region.

In the region of the superior cervical ganglion, synapses are formed with ganglion cells from which the postganglionic fibers arise.[5] The fibers are continued into the skull with the internal carotid artery and, joining the branches of the internal carotid nerve, connect with the cavernous plexus; here they join the ophthalmic division of the trigeminal nerve, and by the nasociliary and long ciliary nerves reach the pupil; other fibers go to the tarsalis muscle, the orbitalis muscle and other muscles. Evidently a lesion of the fibers may occur in any part of their course.

When ocular signs are present the tumor has already invaded the skull.

In the group of patients with ocular symptoms, involvement of the cervical glands was not as common as is usually reported.

Horner's syndrome is a frequent and important sign.

5. Moore, R. F.: Medical Ophthalmology, Philadelphia, P. Blakiston's Son & Co., 1922, p. 95.

CONCLUSIONS

After a study of these cases, with their numerous ocular manifestations, the ophthalmologist must be impressed with the importance of recognizing tumor of the nasopharynx as part of his work.

In any obscure case in which there are pain in the head and involvement of cranial nerves, nasopharyngeal tumor must be considered.

One can evolve an ocular syndrome of nasopharyngeal tumor: Involvement of the fifth, sympathetic and sixth nerves, either individually or together, with symptoms referable to the ear, are strongly suggestive of a nasopharyngeal tumor.

REPORT OF CASES

CASE 1.—J. H., a girl aged 17 years, was first seen in the office on Nov. 30, 1936. She was referred by an otologist and a neurologist. She complained of headaches over the right eye and the right side of the head, which had been present for three weeks and had been almost constant. For two weeks she had occasionally seen double. The condition of the right ear had been treated for two months, with seeming improvement. She had an abscess of the ear lanced twice, but hearing was reduced.

Examination showed vision of 20/20 in each eye. There were slight esotropia for distant vision and esophoria for near vision. The fundi and fields were normal. With tests with light and red glass, homonymous diplopia was found to be present, which increased when the eye turned to the right. The palpebral fissure of the right eye measured 10 mm., and that of the left, 12 mm. The pupil of the right eye was 5 mm. in diameter, and that of the left eye was 6 mm. in diameter. After the instillation of cocaine hydrochloride the right palpebral fissure remained the same, but the left definitely widened. The pupils remained the same. The involvement of the fifth nerve, the paresis of the sixth nerve and an incomplete Horner syndrome, with symptoms referable to the ear, made one think of the possibility of a nasopharyngeal tumor.

The neurologist's diagnosis was tumor of the brain or low grade encephalitis of obscure origin.

The report of the roentgen examination was as follows:

"The inner end of the right petrous pyramid is not as dense as that of the left, and the adjacent portion of the greater wing of the sphenoid bone is somewhat atrophic. The air cells in the right petrous pyramid and the right mastoid process are not as clear as those on the left. There is no evidence of a mass in the nasopharynx, and the lateral view of the cervical portion of the spine fails to show any evidence of a mass in this region. The cervical portion of the spine has lost some of its normal anterior convexity. The findings suggest a tumor involving the floor of the right middle fossa."

The specimen of tissue from the fossa of Rosenmüller was examined but did not show any tumor. However, the mass was probably too small to be reached.

This patient has received high voltage roentgen therapy, with marked improvement of her symptoms.

CASE 2.—R. B., a man aged 52, was first seen in the office on March 30, 1933. The chief complaint was epiphora. The left eye felt sore and "watered" all the time. Hearing in the left ear had been reduced for the past year. Headaches and earache had occurred for four months. Numerous teeth had been extracted. The

patient had an operation on the mastoid process two months previously. Recently he had had difficulty in swallowing, and the voice was hoarse. There was numbness along the left ear and left cheek to the chin.

Examination showed vision of 20/20 in each eye. Orthophoria was present. The fundi were normal. The pupils were unequal, the right being larger than the left; each reacted to light and in accommodation. The palpebral fissure of each eye measured 9 mm. The pupil of the right eye was 3 mm. in diameter, and that of the left eye was 2 mm. in diameter. After the instillation of solution of cocaine hydrochloride the palpebral fissure of the right eye measured 10.5 mm., and that of the left, 9 mm. The right pupil was 6 mm. in diameter, and the left pupil was 2 mm. When examination was made with the exophthalmometer each eye measured 17 mm. There were involvement of the fifth and seventh nerves and Horner's

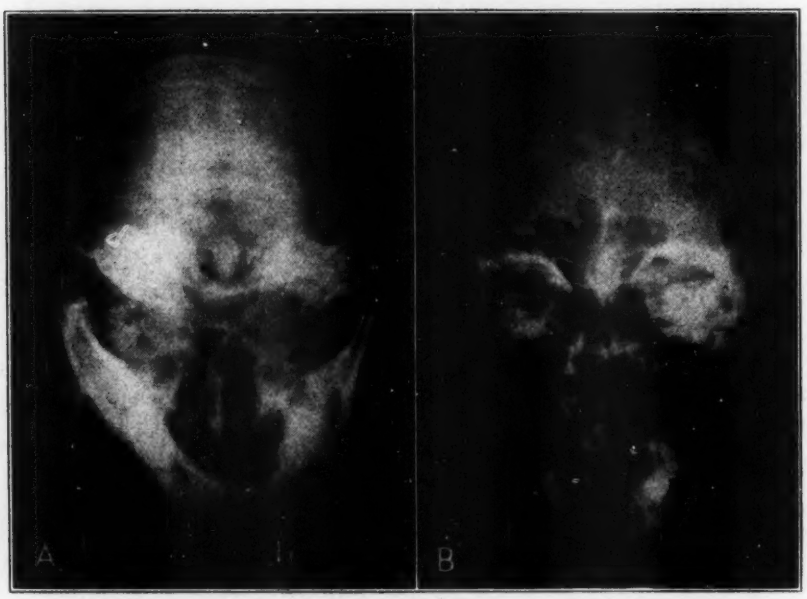

Fig. 1 (case 1).—These roentgenograms illustrate a very early case. There is already involvement of the petrous pyramid and the adjacent portion of the greater wing of the sphenoid bone.

syndrome. The jaw deviated to the right, and there was apparently some involvement of the laryngeal nerves.

The diagnosis was a nasopharyngeal tumor.

Mastoidectomy revealed a normal mastoid process. An outstanding otologist operated, and two prominent otologists saw him in consultation.

A biopsy of tissue from the fossa of Rosenmüller was reported as showing carcinoma. There was a granuloma in the middle ear; this was examined and the pathologic report was the same as the previous one.

The patient gradually became worse. Neuroparalytic keratitis developed. Practically all the cranial nerves on the left side became involved. He had numerous roentgen treatments. He died in November 1935.

Case 3.—A. W., an Austrian housewife aged 54, was admitted to the hospital with a history of headaches for three months and diplopia for two and one-half

weeks. In association with the diplopia convergent strabismus developed. The headaches were worse on the right side and in the frontal region. There had been some pain in and behind the eyes, particularly the right eye, for two and one half weeks. The patient was in the hospital from March 30 to April 18, 1934. The diagnosis was squamous cell carcinoma of the nasopharynx.

Examination showed weakness of the external rectus muscle bilaterally, and unequal pupils, the right being larger than the left. There were no other findings except that the patient was somnolent, lying in bed with her head buried in the pillows most of the time. When spoken to, she complained bitterly of pain. Horner's syndrome was present. The right pupil was 4 mm. in diameter; the left was 3.5 mm. in diameter. The right palpebral fissure measured 8 mm.; the left, 6 mm. Thirty minutes after the instillation of cocaine hydrochloride the right pupil was 6 mm. in diameter, and the left, 3.5 mm. in diameter; the right palpebral fissure measured 10 mm., and the left, 7 mm. With the exophthalmometer each eye measured 16.5 mm. The patient's vision and fundi were normal. Examination of the ears gave negative results.

Numerous diagnoses were made—a cerebral neoplasm or neoplastic inflammatory disease of the meninges, syphilis (to be ruled out by serologic tests), sinusitis, encephalitis, an expanding tumor and a nasopharyngeal tumor.

Examination of the nasopharynx showed a mass of tissue. A biopsy was performed, and a report of medium ripe squamous cell carcinoma was made. A roentgenogram of the skull showed that the sella turcica was normal in size and shape; there was marked decalcification of its floor without definite evidence of destruction. There was marked clouding of the ethmoid sinuses on each side, and of each antrum, particularly the right. The patient received roentgen therapy, without improvement.

CASE 4.—D. L., a man aged 46, a British West Indian porter, three months before admission to the hospital began to notice swellings in his neck, first on the left side and then on the right, which were not painful. Since that time they had remained approximately the same size. They were accompanied by rather sharp pain at the nape of the neck, radiating over the skull to the eyes. Three weeks before admission he expelled a small amount of blood from his throat, at which time he began to be troubled with double vision on looking toward the right. For five days before admission he had a noticeable increase in his dysarthria, with impairment of taste, smell and hearing on the left side. There had been some hyperesthesia under the left lid. He was in the hospital from July 9 to 19, 1931. The diagnosis was carcinoma of the nasopharynx, with metastasis to the glands of the left side of the neck.

Physical examination revealed the left palpebral fissure to be smaller than the right, and the left pupil to be smaller than the right; there was moderate enophthalmos in the left eye. Examination of the neck revealed several soft fused nodes running from the angle of each mandible downward along the posterior cervical chain. There were no other abnormal findings. A mass was removed from the nasopharynx; it was reported to be an unripe squamous cell carcinoma.

CASE 5.—S. L., a 49 year old Chinese man, had been in good health until six months before admission to the hospital, when he began to have difficulty in opening the left eye. Since then there had been gradually increasing inability to move the eyeball, and he had severe headaches in the left occipital region. He had marked anorexia, weakness and loss of weight. He was in the hospital from June 22 to July 27, 1934.

Physical examination showed a cachectic Chinese man. The left pupil was fixed in moderate dilatation. The left eyelid was completely ptosed and could not be raised. There was slight exophthalmos of the left eye, and this eye was practically immobile. Vision was normal. There were small nontender nodes in each inguinal region. The impression of a nasopharyngeal neoplasm with metastases in the region of the sphenoid fissure was confirmed by the finding of neoplastic tissue in the nasopharynx. A piece of it was removed. The pathologic report was lympho-epithelioma (Schmincke tumor). However, the report based on further biopsies was transitional cell carcinoma. A roentgenogram of the skull showed extensive destruction of the inner wall of the left orbit, involving the sphenoid fissure; this process extended into the adjacent ethmoid sinuses. The patient received numerous roentgen treatments to the head, with no improvement. He was transferred to another hospital.

CASE 6.—L. G., a 44 year old married woman, had a past history of tuberculosis, which was detected two years before admission to the hospital, and poor hearing on the left side for seven years. Eleven months before admission she complained of dizziness and nausea, which had continued to the date of admission. Six months before her nasal septum and sinuses were operated on, with slight relief. One month later she began to complain of double vision, convergent strabismus of the left eye, and twitching of the left upper lid. However, before admission she began to complain of intense pain over the left eye and a sensation of numbness over the left corner of the mouth. She was in the hospital from March 10 to May 12, 1931. The diagnosis was lympho-epithelioma of the nasopharynx, with metastases to the base of the skull.

Examination showed: (1) palsy of the left external rectus muscle, (2) Horner's syndrome on the left side, (3) slight asymmetry of the right side of the face, (4) diminution of the abdominal reflexes on the right and an equivocal Babinski sign on the same side, (5) chronic fibroid phthisis and (6) impairment of conduction in each ear, which was more marked on the left side.

Examination of the nasopharynx revealed a mass; the report based on a biopsy was "lypmho-epithelioma."

Operation on the sphenoid sinuses, with the removal of the anterior walls, revealed a dehiscence in the floor of the anterior fossa, overlying the anterior wall of the sphenoid bone. Tissue removed from the sphenoid sinuses was also reported as being lympho-epitheliomatous. The entire neurologic syndrome was explained on the basis of metastasis to the tissues about the pharynx and the base of the skull. The patient was given intensive radiotherapy bilaterally, with some evidence of improvement. On discharge there was some increase in the power of the left external rectus muscle, and there was great diminution of the pain previously complained of. Signs of involvement of the pyramidal tract on the right side had increased, and there were also signs of involvement of the pyramidal tract on the left, indicating a distinct increase in the intracranial pathologic condition.

CASE 7.—D. D., a man aged 53, was admitted to the hospital complaining of pains in the face of one year's duration. Six weeks before admission a radical operation was done on the left frontal sinus. Three years before admission the patient began to have frequent sore throat. One year before admission he began to notice frequent shooting pains in the left gums; several teeth were extracted, but to no avail. Shortly thereafter he noted diminution of auditory acuity on the left side. Eight months later the pains in the left side of the face became

generalized, radiating backward and down the left side of the neck and shoulder, and even down the left arm. A radical operation on the frontal sinus did not relieve the pain. He was in the hospital from April 13 to 27, 1934.

General physical examination showed the scar at the site of the operation on the sinus. The soft palate and uvula were red and edematous; the median raphe was pushed to the right by a mass above the soft palate on the left, and the left submandibular glands were tender. Neurologic examination showed tremor of the hands and tongue and sensory changes in the distribution of the left supraorbital nerve. The pupils were small, the right being larger than the left, and reacted to light and in accommodation. The left palpebral fissure was smaller than the right. There was deafness of the conduction type on the left side. Examination of the pharynx showed several nodules in the nasopharynx. It was thought that these nodules, by occlusion of the eustachian canal and pressure on the sphenopalatine ganglion, accounted for the patient's symptoms. The first biopsy showed hyperplastic lymphatic tissue. The report based on the second biopsy was transitional cell carcinoma. The patient was referred for radiotherapy. Roentgen examination showed destruction of the wall of the antrum, which would lead one to believe that there was a carcinoma of the antrum, with destruction of the contiguous bone.

CASE 8.—G. B., a man aged 34, a musician, was admitted to the hospital on April 19, 1935, for relief of headache in the right frontal region, of seven months' duration. Swelling of the orbits had been present for four months. The personal history was not significant. The family history was irrelevant.

About seven months before admission, photophobia developed, which was somewhat relieved by the wearing of smoked glasses. Shortly thereafter, diplopia appeared. In November 1934 a dull headache in the right frontal region developed, which became almost constant. About one week later the right nostril became plugged, and there was a moderate amount of pain about the inner angle of the right orbit and the upper part of the right side of the nose. This pain was distinct from the headache. Since the nasal obstruction did not clear up, the patient visited a hospital clinic, where, following treatment, he had moderate epistaxis for one week. By the end of December proptosis of the right eye was noted. He was admitted to the Montreal General Hospital on two occasions. There he received radiotherapy. A report from the hospital stated that the exophthalmos receded during treatment. He returned from Montreal four weeks before admission, with bilateral proptosis and ecchymosis. Three and one half weeks before admission he was operated on. The left eye was completely closed, and he could barely see with the right. He complained of constant pain and numbness of the forehead and dull constant pain in the right side of the face. Local examination at that time showed total amaurosis of the left eye and marked impairment of vision in the right eye (counting fingers at 2 feet [60 cm.]). Bilateral proptosis, with edema and chemosis in each eye, and bilateral atrophy of the optic nerve were present. There was marked limitation of ocular movements in all directions. The impression before operation was that of chronic thrombosis of the cavernous sinus or an intrasellar or suprasellar growth. Lumbar tap showed a clear fluid; the initial pressure was 300 mm. of water, and a cell count showed 2 lymphocytes. A roentgenogram of the skull showed marked destruction of the nasal portion and of the adjacent frontal portion of the left orbit, with somewhat less external destruction of the corresponding part of the right orbit. The bridge of the nose also seemed to be involved, and the anterior ridge of the left sphenoid bone was partially destroyed.

An operation was performed on April 20, 1935, at which time small foci over the frontal bone were found to contain pus, and as the operation progressed, pus was found in the bone itself and under the bone, and there was a thick layer of shaggy granulation tissue overlying the dura, particularly in the left frontal lobe. The patient was transferred to the neurologic service on May 8. He was bedridden and disoriented, and had bilateral anosmia and complete blindness. Histologic examination of the specimen removed at operation showed a Schmincke tumor and osteomyelitis. Radiotherapy was considered inadvisable. The patient became progressively worse, and died on May 17.

Fig. 2 (case 8).—Sections of the brain, showing invasion of the frontal lobes by the tumor.

Autopsy showed extensive bony involvement and involvement of both frontal lobes. Besides the anterior portion of the frontal bone, the orbital surfaces of both anterior fossae were involved as far back as the anterior clinoid processes in the midline and almost as far back as the ridges of the sphenoid bone on the sides. The dura was thickened by the tumor.

Microscopic examination showed lympho-epithelioma (a Schmincke tumor) and encephalopathy due to compression.

Case 9.—B. S., a man aged 68, complained of nosebleeds and headaches of three months' duration and of numbness of the left side of the face. Three weeks

before admission he had an operation on the left side of the nose for relief of nasal obstruction due to sinus disease. He complained of difficulty in eating and swallowing, impairment of hearing on the left side, drooping of the left eyelid and drowsiness of three weeks' duration. He was in the hospital from Jan. 27 to Feb. 11, 1927. The diagnosis was carcinoma of the nasopharynx complicated by destruction of the base of the brain.

Examination showed anosmia on the left side. Examination of the fundi showed some sclerosis of the retinal vessels; otherwise it gave negative results. The pupils were slightly dilated and did not react to light or in accommodation, and the corneal reflex on the left side was lost. There was paralysis of the left levator muscle, with weakness of the right levator muscle. There was paralysis of all the ocular muscles of each eye, and sensory paralysis of the left side of the face was also present. The left masseter muscle and the left pterygoid muscle were paralyzed. The function of the left facial nerve was impaired. Taste was lost on the left side of the tongue. Hearing on the left side was much impaired. The glossopharyngeal nerve and the vagus nerve on the left side were paralyzed. There were partial paralysis of the eleventh nerve on the left side and paralysis and atrophy on the left side of the tongue. This condition was diagnosed as a neoplasm in the base of the brain. Roentgen examination showed absorption of bone in the entire middle fossa, the base of the brain and the sella turcica, and the sphenoid sinuses were destroyed. Stereoroentgenograms showed an oblong tumor projecting into the base of the brain from the upper nasopharyngeal region. A specimen from the nasopharynx was reported as showing carcinoma.

CASE 10.—S. S., a man aged 46, was admitted to the hospital with a history of pain in the left ear for one year. He had had several operations done outside the hospital. He had noticed weakening of the left side of the face and inability to close the left eye for seven weeks. The chief complaint was paralysis of the left side of the face, of one week's duration. He was in the hospital from March 29 to April 6, 1927. The diagnosis was carcinoma of the nasopharynx (the left eustachian tube).

Examination showed the right pupil to be larger than the left; each reacted promptly to light. The fundi and vision were normal. There were paralysis of the left side of the face and partial paralysis of the sensory division of the fifth nerve. The tongue deviated to the right. There was bilateral involvement of hearing, which was more pronounced on the left. There was impairment of taste on the left side of the tongue anteriorly and posteriorly.

The following diagnoses were made: (1) an infectious process involving the meninges around the medulla; (2) basilar localized circumscribed meningitis; (3) a disease process in the course of the left seventh nerve in the fallopian canal, due either to involvement of the nerve or to some systemic disease such as diabetes or syphilis.

Examination of the nasopharynx showed a mass in the eustachian tube. The report based on a biopsy was carcinoma.

The otologist reported that the appearance in the left ear indicated a neoplasm rather than a suppurative process.

In December 1928, after radium and roentgen treatment had been given for a long time, the glandular swelling had disappeared. There was no evidence of a growth in the nasopharynx. The deafness had completely disappeared, and there was only a residue of the signs due to the original involvement of the seventh nerve. Roentgenograms of the accessory sinuses were entirely negative. The patient might

be said to be free from disease. However, on Feb. 18, 1931, a social worker notified the department that he had died at home.

CASE 11.—R. L., a 54 year old widow, was admitted to the hospital on April 9, 1934. Six months before admission she began to complain of severe headache in the occipital region. This occurred practically every night and had continued to the date of admission. Four months before admission she noticed a sensation as though she had hair on the right side of the mouth. Three months before admission she felt that the right side of her face was frozen and the inside of her mouth was numb. Associated with these complaints was severe shooting, stabbing pain about the right side of the face, often localized in the right eye and associated with lacrimation. For two months she had noticed roughening and thickening of the right side of the face, especially about the nose. For six weeks she had a burning sensation of the entire lips and the right side of the tongue. She was in the hospital from April 9 to May 7, 1934. The diagnosis was carcinoma of the nasopharynx. Lumbar tap and radiotherapy were carried out.

Examination revealed massive hard lymph nodes on the sides of the neck with enlargement of the axillary nodes. There was questionable irregularity of the posterior pharyngeal wall. She presented Horner's syndrome on the right: ptosis, myosis and enophthalmos. With the cocaine test the left pupil dilated to a size three times greater than that of the right. There was anesthesia of the right cornea. There was involvement of all the modalities of sensation in all the divisions of the right fifth nerve. It was felt that the involvement of the fifth nerve was due to a malignant disease in the vicinity of the gassarian ganglion. Examination of the ears, nose and throat revealed a small pea-sized flat purplish mass in the nasopharynx, with superficial erosion. Pathologic examination was reported to show an embryonal carcinoma (the so-called Schmincke tumor). Treatment consisted in radiotherapy. Roentgen examination of the skull and paranasal sinuses showed a large mass in the floor of the left antrum and slight clouding of the sphenoid sinuses but no definite erosion of the wall of the sphenoid sinus. The sella turcica was moderately enlarged.

On August 14, 1935, the patient died.

CASE 12.—J. E., a 52 year old Irish laborer with a history of two previous admissions for squamous cell carcinoma of the soft palate and nasopharynx, was in the hospital from Oct. 25 to Nov. 3, 1934. The diagnosis was squamous cell carcinoma of the pharynx, with secondary carcinoma in the base of the brain.

The patient was treated with radiotherapy. There had been attacks of frequency of urination, urgency and burning for ten months, an attack of unconsciousness and clonic convulsions with fecal incontinence one month before admission and frequent attacks of dizziness, nausea and momentary loss of vision for the week preceding admission. There had been diminution in smell for one week before admission.

Examination showed that the entire soft palate was missing. There were reddish elevations along the pillars of the tonsillar fossa.

Neurologic examination showed slight past pointing with the left hand, a fine tremor of the outstretched hands and generalized hyperreflexiae. There were no pathologic reflexes, no motor weakness and no sensory abnormalities. Signs of involvement of the cranial nerves were anosmia on the left and diminished smell on the right. The left pupil was smaller than the right. The left lateral wall of the palate did not move as well as the right; there were dysphagia, atrophy of the left half of the tongue and deviation of the tongue to the left when protruded.

The findings and history pointed to diffuse metastatic involvement of the brain, probably to the base. The impression was that of carcinomatous metastasis to the base of the skull.

CASE 13.—A. F., a woman aged 51, was first admitted to the hospital on July 1, 1929, remaining until July 30. The diagnosis was neuralgia (the sphenopalatine nerve being affected) and sinusitis. Lumbar puncture was done, and the condition improved.

She was admitted a second time, remaining in the hospital from April 28 to June 4, 1930. The diagnosis was transitional cell carcinoma tumor of the brain (metastatic).

The patient began to have pain in the left ear two years before admission and was treated for sinus disease. She had a postnasal discharge following a radical operation on the antrum in December 1928. The pain became worse, and deafness developed in the left ear. In July 1929 the patient was in the Mount Sinai Hospital, where a diagnosis of neuralgia of the sphenopalatine nerve on the basis of sinusitis was made. In October 1929 the purulent discharge became severe, and examination revealed a mass near the spheno-ethmoid region. A biopsy was performed, and the tissue was diagnosed as showing carcinoma. The patient was given four implantations of radium in December 1929. She returned because of persistent pain, a "smoky" taste on the tongue and diplopia. The last symptom came on one month previously. Examination showed weakness of the left external rectus muscle, diplopia, weakness of the left motor trigeminal nerve, involvement of sensation in the second and third branches of the left trigeminal nerve, and loss of taste on the anterior two thirds of the tongue on the left side. There was diminished aerial conduction on the left side, with tinnitus and thickening of the left ear drum. Caloric tests showed a normal response. The fundi were normal. Spheno-ethmoidectomy was performed on the left, and the material was examined. It was reported as showing transitional cell carcinoma. It was felt that the condition was a primary tumor of the nasopharynx which was eroding the roof of the ethmoid bone and growing along the base of the skull. Radium seeds were implanted in the nasopharynx, and shortly after this the patient lost the vision in the left eye. The left pupil became fixed to light, and there was some photophobia. There was little improvement in the power of the left side of the face. Further roentgen treatment was advised.

On the third admission the diagnosis was tumor of the brain (the middle fossa)—transitional cell carcinoma. The patient was in the hospital from Dec. 22 to 28, 1930. An injection was made into the supra-orbital and infra-orbital nerves on the left side, with some relief. There was some exophthalmos of the left eye following the injection, probably due to secondary edema.

The patient died at the New York City Cancer Institute Hospital on April 3, 1931. No autopsy was performed.

CASE 14.—A. K., a man aged 47, was in the hospital from July 11 to 20, 1927. The diagnosis was lymphosarcoma of the nose and sinuses. His condition was unimproved.

The chief complaint was headache over the left eye. There was exophthalmos, and the left eye turned out.

From eight to ten months before admission headaches gradually developed. These were mostly over the left eye. There was also pain in the left ear and over the right antrum. Protrusion and turning out of the left eye were also present, which symptoms had persisted. Although the patient had had three operations on

the inside of the left side of the nose at another hospital in March, May and June 1927, the nose on that side was blocked.

On admission he had proptosis and papillitis of the left eye. The eye was pushed out and forward, and there was no inward rotation beyond the midline. There was a mass palpable in the floor and on the nasal side of the orbit. A specimen removed from the region of the left middle turbinate showed lymphosarcoma. The patient was referred for radiotherapy.

He was readmitted to the medical service of the Mount Sinai Hospital on April 5, 1929. There were metastases to the small intestines. He was referred to the Montefiore Hospital, to which he was admitted on May 22. He died on June 4.

Postmortem examination confirmed the clinical diagnosis of lymphosarcoma of the nasopharynx and metastases to the jejunum, with perforation.

CASE 15.—J. L., a rabbi aged 52, was admitted to the hospital complaining of double vision of seven months' duration and pain in the left side of the head of four months' duration. He had had diabetes for ten years, which had been treated by diet and insulin. Seven years before admission the tonsils were removed because of frequent sore throat. The patient was well up to seven months before admission, when diplopia suddenly developed, which persisted up to the time of admission. Four months before admission nasal obstruction developed, without any symptoms of cold. The obstruction was more marked on the right side. On several occasions the discharge from the left side of the nose was blood tinged, and two months before admission there was frank bleeding. About three and one half months before admission the patient noticed progressive diminution of hearing in the left ear. Following that he noted a small hard nodule in the region to the left of the vertex, with some itching and with pain radiating to the left frontal region and the left side of the head, face and neck. Though all his teeth had been removed two years previously, he had pain in the left upper and lower jaw. About three months before admission the left side of the face and the left side of the tongue felt numb and frozen. Eight weeks before admission a painless, hard swelling appeared on the left side of the neck, which for a time increased in size. For the past six weeks he had become considerably weaker. He was in the hospital from May 8 to 29, 1935. The diagnosis was carcinoma of the nasopharynx and diabetes mellitus. Radiotherapy was given, and a biopsy of tissue from the nasopharynx was done.

Physical examination showed a small nodule about 4 mm. in diameter on the left of the midline over the vertex. There was a large hard mass under the left angle of the jaw, 3 cm. in diameter, nontender and fixed to the underlying tissue. Neurologic examination showed anosmia bilaterally and paresis of the left external rectus muscle, and the corneal reflex was diminished on the left. There was hyperalgesia over all three branches of the left trigeminal nerve. There was deafness due to involvement of the middle ear on the left side. The soft palate was reddened and possibly pushed forward.

Pharyngeal examination revealed a large ulcerating mass at the roof of the nasopharynx; this was more marked on the left side. A specimen removed from the region was reported as showing embryonal cell carcinoma.

The patient was treated with radiotherapy.

Roentgen examination of the skull showed partial erosion and considerable thinning of the floor of the sella turcica. Following radiotherapy the glands in the neck diminished in size, but the neurologic status was unchanged.

The fundi and fields were normal.

The last follow-up noted was as follows: "The patient still has pain in the left side of the head. There is no difficulty with hearing. There are no gross neurologic evidences of disease. The presence of pain in the left side of the head suggests possible cranial involvement."

CASE 16.—This case was reported in the ARCHIVES.[6] The patient was J. C., a man aged 36.

Numerous biopsies were done, and reports were negative for tumor. Finally, on Feb. 18, 1933, tissue from the nasopharynx and from the anterior wall of the sphenoid bone was reported as showing transitional cell carcinoma.

Numerous complications of the brain developed, and the patient became very anemic and emaciated. He died at the Montefiore Hospital.

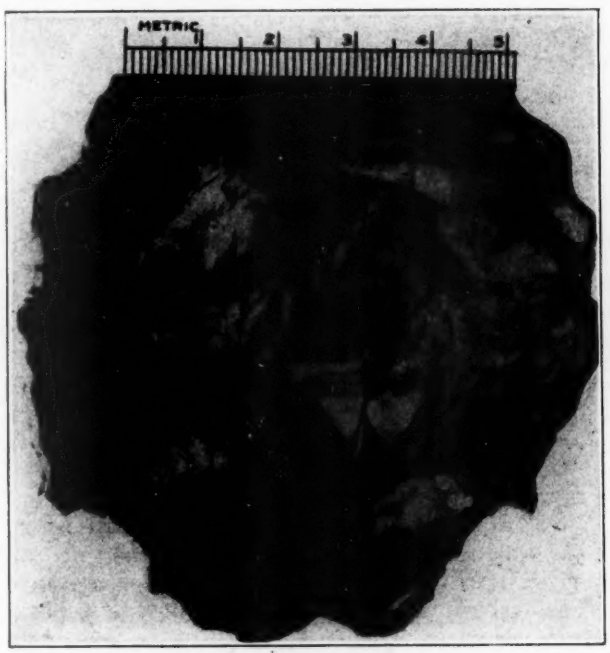

Fig. 3 (case 16).—Photograph showing the tumor involving the entire sphenoid bone.

Postmortem examination was done on November 7. The tumor involved the entire sphenoid bone, spreading laterally and filling up all the basilar foramina, involving the cranial nerves at their exit through the foramina. The tumor compressed these structures: the olfactory nerves, the optic nerves and chiasm and all the cranial nerves, those on the left side being affected more than those on the right. On the right the fifth, sixth and seventh cranial nerves were spared. Part of the brain was destroyed, and the tumor invaded the left temporal convolutions. Microscopic examination showed carcinoma of the sphenoid bone, with cerebral invasion.

6. Schlivek, K.: Schmincke's Tumor, Arch. Ophth. **7**:928-929 (June) 1932.

DISCUSSION

Dr. William Needles: Recently I had occasion to review practically the same series of cases which Dr. Schlivek has reported, but from a different angle, from the neurologic standpoint. There is little I can say to an ophthalmologic audience on the ocular complications of nasopharyngeal tumors, but I wish to mention a point or two.

Of the thirty-eight patients reported on, only one had papilledema, and I think that is readily understood if one realizes the manner of growth of these tumors. While they invade the base of the skull, they grow so as not to cause increase in the intracranial pressure. They push the dura up before them and cause pressure on the nerves in their extradural portion; hence the relative rarity of papilledema or any other evidence of increased intracranial pressure.

As regards the mechanism for Horner's syndrome, there is no single unitary explanation; sometimes this syndrome may be due to pressure on the enlarged glands of the neck, but at other times it is due to pressure of the tumor itself on a portion of the sympathetic nervous system which accompanies the trigeminal nerve, the so-called paratrigeminal portion of the sympathetic nervous system.

It is important that this subject has again been brought up, because, although numerous contributions have appeared on the subject of nasopharyngeal tumors, I think that the general practitioner, as well as physicians in the various specialties, is still not sufficiently "nasopharyngeal tumor minded" to avoid the mistakes which occur. That point can best be brought out if I cite some of the statistics in the experience of my associates and me. We reviewed thirty-five cases of nasopharyngeal tumor and found that twelve of the patients had had thirty operations performed; two had had tonsillectomies; four had had extraction of teeth; in one case an incision of the gum was made; there were seven myringotomies and one craniotomy; in one case injection into the sphenopalatine ganglion was done; in one case injection into the maxillary nerve was carried out, and one patient had no fewer than six operative procedures before the correct diagnosis was established. The reason for these errors in diagnosis is evident. The patient who has a local symptom, such as ulceration, bleeding or nasal obstruction, is relatively fortunate, because the symptoms point to the true site of the growth; but more often than not the symptoms and signs are referable to distant organs. For example, I found in these thirty-five cases that twenty-four patients had enlargement of the cervical glands, and in seventeen cases that was the first warning signal. One is accustomed to think of tuberculosis, leukemia or Hodgkin's disease as a possible explanation for enlarged cervical glands, but I think it occurs to relatively few physicians to suspect disease of the nasopharynx. In seventeen of thirty-five of these cases, signs referable to the middle ear were present, such as tinnitus, impairment of hearing and a sense of fulness. Attention is usually directed to the middle ear rather than to the nasopharynx. A third indication of these tumors, that leads one away from the nasopharynx, is evidence of involvement of the nerves at the base of the brain. I found in this series, and so have other observers, that the fifth, the sixth and the third nerve are the most frequently involved —in that sequence. In view of this, it would be well in any case in

which there is involvement of the cervical glands signs referable to the middle ear or evidence of involvement of the nerves at the base of the brain to order inspection of the nasopharynx. It is a relatively simple procedure, much less harmful than any of the operative interventions which I have just mentioned.

There is one final point that I should like to make: One need not be frightened away from the diagnosis of a nasopharyngeal tumor by reason of negative results of biopsy. Diagnosis based on biopsy depends on the skill of the observer. But even when the observer is skilled the first specimen may be returned as showing negative results, whereas a second specimen will show positive results. At the neurologic service of the Mount Sinai Hospital it is the rule, even if the biopsy gives negative results, if the symptoms and signs are sufficient to suggest a nasopharyngeal tumor, to institute radiotherapy. In one such case positive results of biopsy were obtained only after radiotherapy had been instituted.

DR. JOHN M. WHEELER: In examining a rather large series of cases Dr. Schlivek has seen more instances of Horner's syndrome than most of the members have seen, and I should like to ask a question. Was enophthalmos a common manifestation in the cases of Horner's syndrome, and, if so, in what proportion of them?

DR. MARTIN COHEN: I should like to report the case of a patient who died three or four weeks ago with a very large mass in the nasopharynx; biopsy failed to show carcinoma. Associated with the growth, which clinically appeared to be a carcinoma involving the nasopharynx unilaterally, the patient had marked exophthalmos, which was present first on one side. This was the first ocular symptom my associates and I noted; then followed complete ophthalmoplegia, with involvement of the trigeminal nerve. Two months later the other eye became involved with the same ocular symptoms—marked exophthalmos followed by complete ophthalmoplegia. Dr. Faulkner saw the patient, and we made a diagnosis of carcinoma involving the nasopharynx and gave a very grave prognosis. The patient was about 65 years of age, and I observed him for five months. The result of roentgen treatment was nil. He finally died, but before his death we made another biopsy of the mass in the nasopharynx, and this showed typical carcinoma. This is in accord with what Dr. Schlivek said; the first biopsy of the tumor gave negative results, and it was only after the patient received roentgen treatment that we were able to make a diagnosis of carcinoma. In this case the cardinal symptom was marked exophthalmos in both eyes rather than any evidence of aural manifestations or enlargement of the cervical glands. This was the first case I have seen of nasopharyngeal carcinoma penetrating the inferior fissure of the orbit and extending into the orbit. Similar involvement occurred in the opposite eye. That lesion was an extension from the nasopharynx.

DR. KAUFMAN SCHLIVEK: Dr. Wheeler's question is an interesting one. Not all of these patients had exophthalmometer readings; more than half of them did, and none of them showed enophth[illegible]os with the actual reading with the exophthalmometer. Some years ago Dr. Lilienthal removed the superior cervical ganglion in numerous cases of so-called pseudo-angina, and typical Horner's syndrome developed. I

tested the whole series with the exophthalmometer before and after the operation. None of them showed enophthalmos. Have you found true enophthalmos, Dr. Wheeler?

DR. JOHN M. WHEELER: No.

DR. KAUFMAN SCHLIVEK: Dr. Cohen remarked about the lateness in finding the tumor in his case. The patient with the enlarged glands in the neck whom my associates and I saw was in the hospital on numerous occasions because he showed new complications all the time. Six biopsies were done, and the tumor was not reported as lympho-epithelioma until almost three years after the original diagnosis was made. He had numerous treatments with the roentgen rays and radium. My associates and I have seen patients with exophthalmos and one patient with a mass in the orbit, but those conditions are rather late manifestations of the disease, although one patient came in with the original complaint of exophthalmos. That condition progressed until complete ophthalmoplegia interna and externa developed.

REPAIR OF RUPTURE OF THE WOUND AFTER EXTRACTION OF CATARACT

REPORT OF CASES

LOUIS BOTHMAN, M.D.
CHICAGO

Rupture of the wound may occur at any time after extraction of a lens. The mishap occurs most frequently on the fourth or fifth postoperative day. It may be the result of a severe spell of sneezing or coughing, a blow by the patient himself, straining, squeezing of the lids or accidental pressure on the globe when the dressing is being done. It may be caused by a slight or severe hemorrhage or may even happen without any apparent cause.

While I was visiting the Second Eye Clinic in Vienna in 1926, Docent Guist showed me six patients in the same ward in which rupture had developed on the fourth day after operation, and he was almost frantic in his search for the cause, which was never determined.

During the 1932 season at Shikarpur Sind, India, rupture of the wound occurred in twenty-one of 1,004 cases.[1] Eleven of these patients had prolapse of a portion of the iris, and ten had an everted cornea, i. e., the sectioned half of the cornea was bent forward, exposing the iris and vitreous, which did not prolapse.

When a wound due to extraction of a cataract ruptures, the corneal lip is elevated, being pushed forward by the prolapsed portion of the iris, the vitreous or a hematoma. There is usually a horizontal or linear groove across the cornea between the lower angles of the corneal section. If nothing is done or if only the prolapsed tissue is excised, a high degree of astigmatism results. A horizontal linear opacity corresponding to the line or groove in the cornea further interferes with vision.

To prevent the formation of an opacity from such a groove and to reduce greatly or prevent a high degree of astigmatism, the following procedure is recommended.

Elschnig [2] described the same operation for prolapse without enlarging the incisions. With his method of repair a high degree of astigmatism remains.

From the Division of Ophthalmology, the University of Chicago, Dr. E. V. L. Brown, Director.

1. Bothman, L.: Illinois M. J. **62**:243 (Sept.) 1932.

2. Elschnig, A.: Augenärtzliche Operationslehre, in von Graefe, T., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, Berlin, Julius Springer, 1922, vol. 2, p. 1093.

TECHNIC

A conjunctival incision is made at the limbus from 4 to 8 o'clock, and the conjunctiva is freed from the upper half of the bulb. This should be done carefully, without pressure on the bulb. A subconjunctival injection with either procaine hydrochloride or physiologic solution of sodium chloride makes this dissection easier. The conjunctiva should be loose enough to cover the entire cornea without being too taut. Sutures should be placed opposite 10 and 2 o'clock above and fixed in the bulbar conjunctiva about 4 or 5 mm. below 4 and 8 o'clock.

A spatula should be introduced into the wound to separate the lips at both lower angles. The corneal section should then be enlarged for from 1 to 1.5 mm. below the horizontal groove. The prolapsed portion of the iris, if not easily replaced, should be excised, and any hematoma should be removed. The remainder of the incision should be reopened with an iris spatula and the cornea stroked into proper position and held there while the conjunctival flap is pulled down from above and the sutures are tied.

As a rule, when the conjunctiva retracts or when the sutures are removed a week later, the wound is smooth, and the cornea is clear and of normal curvature. Only two of the many cases in which the wound was treated by this method are cited to show the results one may expect.

REPORT OF CASES

CASE 1.—C. B., aged 64, who was seen in consultation, had been operated on for a mature senile cataract in the right eye five days before. There had been no complications. On the fourth night following the operation he had a sudden pain, which lasted for about an hour. On the following day, the corneal lips were elevated from 2.5 to 3 mm. The portion of the iris between 10 and 2 o'clock was prolapsed, and its lower border was pulled up almost to the center of the cornea. There was a 0.5 mm. horizontal groove in the cornea from 9:30 to 2:30 o'clock.

The repair operation already described was done, and a large piece of iris was removed. The postoperative course was uneventful, and four months later after a needling vision of the right eye was 10/10 + 3 with a 12.00 D. sphere + 2.00 D. cylinder, axis 170.

CASE 2.—Miss P. M., aged 36, had been operated on for a cataract in the left eye, due to dinitrophenol. The operation was a capsulotomy extraction through a round pupil. On the third night after the operation the resident physician called me up to say that the patient had an expulsive hemorrhage. Because the bleeding and pain had subsided within ten minutes, I did not see the patient until the following morning. At that time the corneal lips were elevated 2.5 mm. Beneath the conjunctival flap was a reddish black elevated mass which appeared to be a hematoma or a portion of the iris. There was a 0.75 mm. groove in the cornea between 9:30 and 2:30 o'clock.

The repair operation was done thirty-six hours after the rupture, according to the technic already described. The black mass was a hematoma, not a portion of the iris. Only a very small bead of iris at 1 o'clock had prolapsed, and this

was replaced. The incision healed promptly. A minute anterior synechia remained at 1 o'clock. The pupil was slightly oval. After a needling six weeks later vision of the left eye was 15/10—2 with a + 10.50 D. sphere + 3.00 cylinder, axis 180. The other eye was operated on later, without mishap.

COMMENT

This technic is not original with me, for I saw Dr. W. I. Lillie use it when we were in India. The results with it are so uniformly good that I wish to bring it to the attention of those who may not have had the opportunity of seeing the operation performed.

122 South Michigan Avenue.

DIAGNOSTIC SIGNIFICANCE OF EPINEPHRINE INSTILLED INTO CONJUNCTIVAL SACS

LOUIS HUBERT, M.D.
Junior Surgeon, the Manhattan Eye, Ear and Throat Hospital
NEW YORK

There is a general belief among ophthalmologists [1] that the instillation of epinephrine hydrochloride into the conjunctival sacs causes dilatation of the pupils. This is contrary to the findings of Loewi,[2] one of the winners of the Nobel prize in medicine and physiology in 1936. He has shown that when epinephrine hydrochloride is instilled into the conjunctival sacs of normal persons no mydriasis will ensue. However, if the sympathetic nervous system is rendered hyperirritable, as may occur in persons with exophthalmic goiter or diabetes, dilatation of the pupils will become apparent. In applying this test to many hundreds of patients in the Manhattan Eye, Ear and Throat Hospital during the last eighteen years, I have found the observations of Loewi to be correct. Moreover, I have noticed other conditions that may produce unilateral hyperirritability of the sympathetic nervous system of the eye and unilateral dilatation of the pupil after the instillation of epinephrine hydrochloride. This phenomenon has a certain diagnostic significance which will be discussed in this paper. In order to understand this subject clearly it will be necessary to review briefly the most essential facts of the vegetative nervous system in general and the sympathetic nervous system of the eye in particular.

GENERAL CHARACTERISTICS OF THE VEGETATIVE NERVOUS SYSTEM

The vegetative nervous system is an outflow from the cerebrospinal nervous system, namely, from the midbrain, medulla and spinal cord. Certain cells have moved out from these areas and formed in different parts of the body a collection of cells or vegetative ganglions, which act as distributing centers. These ganglions retain their connection with the central nervous system by nerve fibers, which are called preganglionic. The preganglionic nerves, therefore, are fibers that connect the central

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Read before the Section of Ophthalmology of the New York Academy of Medicine, Dec. 21, 1936.

1. Berens, Conrad: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 949.

2. Loewi, Otto: Ueber eine neue Funktion des Pankreas und ihre Beziehung zum Diabetes mellitus, Arch. f. exper. Path. u. Pharmakol. **59**:83, 1908.

nervous system with the vegetative ganglions, and in doing so they take part of their course in voluntary motor nerves. From the vegetative ganglions other fibers are given off, which are called postganglionic. The latter are nerve fibers that run from the ganglions to the structures they supply and reach them by taking their course either within sensory nerves or by following blood vessels. It is also well to remember a physiologic principle worked out by Langley,[3] namely, that the preganglionic fibers are interrupted in only one ganglion, although they may pass through several such ganglions.

These characteristics apply to the entire vegetative nervous system, which consists of two divisions: (1) the sympathetic system proper and (2) the parasympathetic system. The first system originates in the cells of the lateral horns of the spinal cord and is directly connected with the sympathetic cords. The second system is not connected with the sympathetic cords and takes its origin, so far as the eye is concerned, in the midbrain and medulla. The latter system will not be considered, as epinephrine has practically no effect on it.

THE SYMPATHETIC NERVE SUPPLY OF THE EYE

In tracing the sympathetic nerve supply to the eye, one should ask oneself: 1. In what part of the central nervous system do the preganglionic fibers originate? 2. Through what motor nerves do these fibers take their course? 3. In what collection of cells or ganglions are these fibers interrupted? 4. Through what sensory nerves or blood vessels do the postganglionic fibers reach the structures they supply?

The sympathetic preganglionic fibers to the eye have their origin in the lateral horn cells corresponding to the eighth cervical and the first and second dorsal segments of the spinal cord, take part of their course within the first and second thoracic motor nerves, enter the sympathetic cord, run through the stellate, inferior and middle cervical ganglions without being interrupted there and finally reach the superior cervical ganglion, where the interruption takes place. From this ganglion postganglionic fibers are given off, which pass to the various structures of the eye by taking their course along certain blood vessels or within certain sensory branches of the fifth cranial nerve (fig. 1).

I wish particularly to call attention to the fact that the vasomotor fibers of the eye and the pupillary dilator fibers run in separate paths. The postganglionic fibers from the superior cervical ganglion, which are destined to become the vasoconstrictor fibers of the eye, continue from the carotid plexus to the cavernous plexus, and from here, by means of the so-called sympathetic root, run through the ciliary gang-

3. Langley, J. N., in Schäfer, E. A.: Textbook of Physiology, New York, The Macmillan Company, 1900, vol. 2, p. 621.

lion without being interrupted and are distributed by means of the short ciliary nerves to the blood vessels of the eye. Anatomically, the sympathetic fibers can be traced to the uveal vessels and also, through a fine

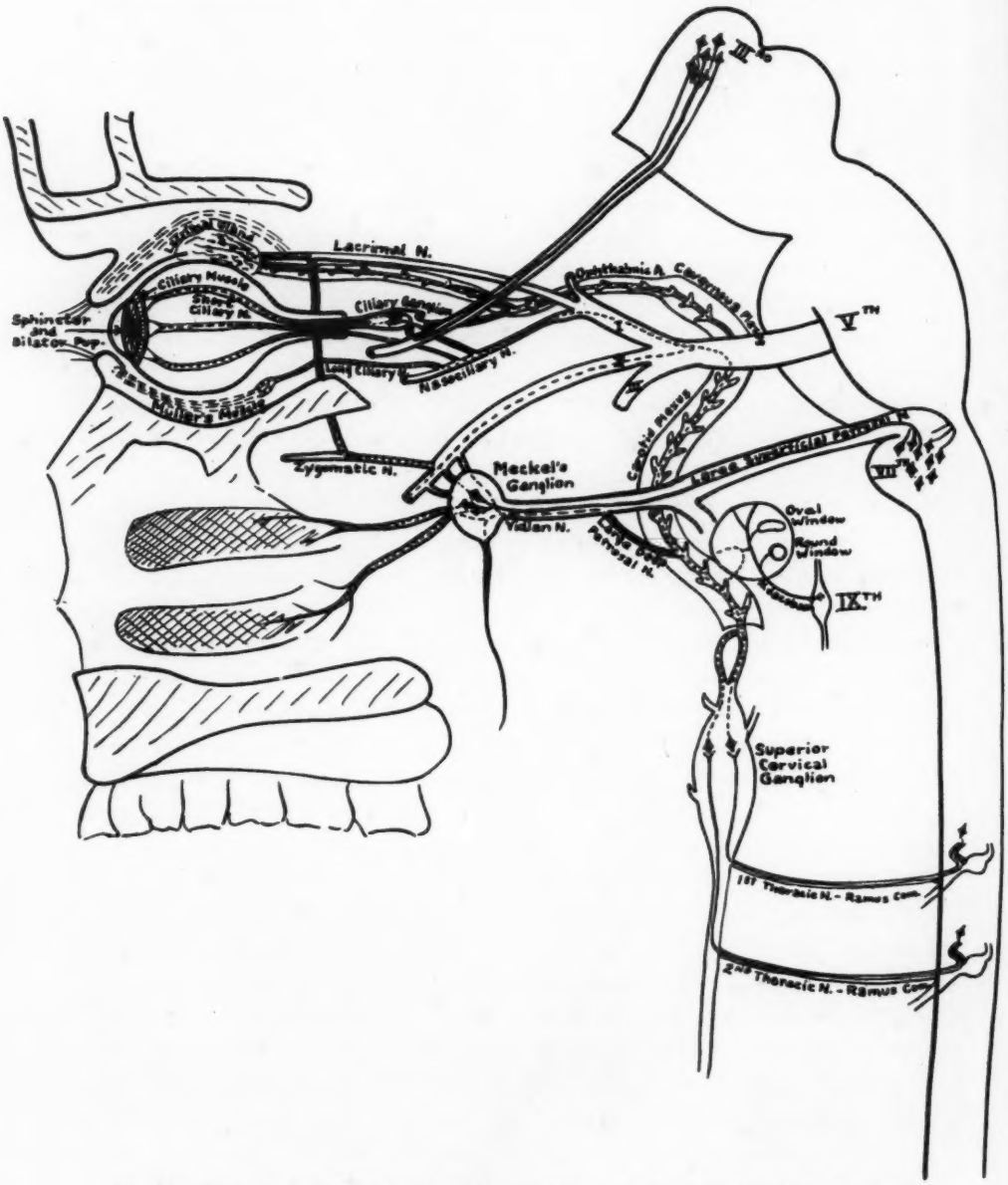

Fig. 1.—The sympathetic and parasympathetic nervous systems of the eye.

sympathetic plexus around the central retinal vessels piercing the optic nerve, to the vessels of the retina. Physiologically, their constricting effect on the uveal vessels, when stimulated by epinephrine, is unde-

niable, but their precise effect on the retinal circulation has been questioned, and there are many reports of contradictory results in the literature.[4]

The course of the postganglionic fibers which supply the dilator muscle of the pupil has been determined by combined physiologic, anatomic and embryologic studies of many investigators.[5] After leaving the superior cervical ganglion they follow the internal carotid artery for a short distance; then, at the level of the middle ear, some filaments detach themselves and traverse the anterior wall of the middle ear to reach the promontory. One of these filaments, at least, joins the tympanic plexus, but others frequently rejoin the carotid plexus after they have traversed the tympanic cavity. The majority of the fibers continue along the carotid plexus, enter the skull with the carotid artery, travel over the gasserian ganglion, accompany the first division of the fifth cranial nerve and its nasociliary branch and join the long ciliary nerves and their branches which enter the eyeball in company with the long posterior ciliary arteries, thus avoiding the ciliary ganglion. The branches of the long ciliary nerves run forward in the epichoroidal space and enter the ciliary body, whence they reach the dilator muscle of the iris.

The postganglionic fibers supplying the bundles of smooth muscles in the upper and the lower eyelids and the orbital muscle of Müller also take their course in some of the branches of the long ciliary nerves.

ACTION OF EPINEPHRINE ON DENERVATED SMOOTH MUSCLES OF THE EYE

Much of the present knowledge of the sympathetic nervous system has been aquired through the application of various drugs. Epinephrine is one of these drugs. In order to understand its effect on the pupil, as well as on the smooth muscular fibers of the lids and Müller's orbital muscle, it is well to remember a fact long known to physiologists and only recently appreciated by neurosurgeons, namely, that denervated smooth muscles become sensitive to the circulating epinephrine in the blood, and of course will become still more sensitive when this substance is directly applied by conjunctival instillation. If the dilator muscle of the pupil and the other bundles of smooth muscles of the eye are cut off from their nerve connection with the superior cervical ganglion (i. e., if the lesion is in the ganglion or its postganglionic fibers), epinephrine hydrochloride instilled into the conjunctival sac will act

4. Duke-Elder, W. Stewart: Text Book of Ophthalmology, London, Henry Kimpton, 1932, p. 415.

5. Piquet, J., and Coulouma, P.: Les signes oculo-sympathiques dans les otites et leurs raisons anatomiques, Ann. d'oto-laryng., September 1935, p. 1003.

directly on the denervated muscle fibers and cause not only dilatation of the pupil but elevation of the lid and slight protrusion of the eyeball.

In the clinics of the Manhattan Eye, Ear and Throat Hospital my associates and I often find patients with paralysis of the cervical sympathetic system resulting in Horner's syndrome. The lesion causing this condition may be in the superior cervical ganglion, the preganglionic and postganglionic fibers or the central nervous system. That there is such a paralysis can be determined by the obvious physical signs and by the absence of dilatation of the pupil following the instillation of a 4 per cent solution of cocaine hydrochloride into the conjunctival sac. Usually it is easy to locate the lesion causing the syndrome. Occasionally the instillation of epinephrine hydrochloride into the conjunctival sac may be the only means of localizing it. It will tell one with a reasonable degree of certainty whether the lesion is (1) in the superior cervical ganglion and its postganglionic fibers or (2) in the preganglionic fibers and the central nervous system. In the first instance there results denervation of the smooth muscles of the eye supplied by the sympathetic nervous system and hypersensitiveness to epinephrine. In the second instance no denervation and no hypersensitiveness follow. To illustrate these points a few cases are reported.

CASE 1.—L. F., a 52 year old woman, came to the clinic on July 22, 1932, for examination of her eyes. She had a complete Horner syndrome on the right side, of eighteen years' duration. Five years before she came to the clinic a tumor on the right side of her neck, at the level of the superior cervical ganglion, was removed. The nature of the tumor is not known. Its removal had no effect on her eye. The instillation of a few drops of a 4 per cent solution of cocaine hydrochloride into the conjunctival sac of each eye produced dilatation of the left pupil, but the right pupil remained constricted (fig. 2 *A*). The instillation of epinephrine hydrochloride in the strength of 1:1,000, however, resulted not only in greater dilatation of the left pupil but in dilatation of the right pupil. Moreover, the drooped right lid lifted itself up, and the enophthalmos and the narrowing of the slit of the lid of the right eye temporarily disappeared (fig. 2 *B*). This test was repeated three times at variable intervals, with the same results. The lesion in this particular case evidently involved either the superior cervical ganglion or its postganglionic fibers, or both.

CASE 2.—M. S., a 15 year old boy, came to the clinic on March 13, 1935, complaining of sore throat, difficulty in opening the mouth and stiffness of the neck on the right side. Examination revealed a swelling in the right lateral pharyngeal wall. A diagnosis of a parapharyngeal abscess was made. An incision into the swelling resulted in severe hemorrhages from the throat. The right external carotid artery and the ascending pharyngeal arteries were tied off. The hemorrhages continued from the throat and also occurred from the right ear. The right common carotid artery was then ligated in the inferior carotid triangle (the triangle of necessity). I selected this region because of the previous operation and the infection of the neck in the superior carotid triangle. During the last operation I must have injured the preganglionic sympathetic fibers of the right eye, because within a few days Horner's syndrome developed on the right. The

instillation of a 4 per cent solution of cocaine hydrochloride into the conjunctival sac of each eye produced dilatation of the left pupil but not of the right pupil. The latter, as well as the drooped lid and the enophthalmos, remained unaffected after the repeated instillation of epinephrine hydrochloride (fig. 3). This was due to the fact that the superior cervical ganglion and its postganglionic fibers to the smooth muscles of the eye were intact, and therefore no hypersensitivity to epinephrine developed. With the exception of Horner's syndrome, no ill effects followed the ligation of the common carotid artery, and the patient is now in perfect health.

CASE 3.—R. W., a 55 year old man, was seen on Nov. 19, 1936, complaining of drooping of the left upper eyelid and severe paroxysmal pain on the left side of his face. This pain was radiating toward the left ear and the left eye. The trouble started a year and a half previously. The patient showed a complete

Fig. 2 (case 1).—*A*, photograph showing dilatation of the left pupil, with constriction of the right pupil, after the instillation of a 4 per cent solution of cocaine into the conjunctival sac of each eye. *B*, photograph showing dilatation of each pupil, which is greater in the left pupil than in the right, lifting of the right lid and absence of enophthalmos and of narrowing of the slit of the lids of the right eye, after the instillation of epinephrine hydrochloride in the strength of 1:1,000 into the conjunctival sac of each eye.

Fig. 3.—Photograph showing dilatation of the left pupil, and drooping of the lid and enophthalmos of the right eye, after the instillation of a 4 per cent solution of cocaine and the repeated instillation of epinephrine hydrochloride into the conjunctival sac of each eye.

Horner syndrome on the left. The location of the lesion causing the syndrome could not be determined, in spite of a thorough general and neurologic examination, including roentgen studies. The instillation of epinephrine hydrochloride into the conjunctival sac produced not only dilatation of the constricted left pupil but elevation of the drooped left upper lid. The lesion must therefore have been in the postganglionic sympathetic fibers to the left eye.

As the patient complained of severe paroxysmal neuralgic pain characteristic of trifacial neuralgia and as the postganglionic fibers to the pupil and the lids passed over the gasserian ganglion (fig. 1), may one not assume that the lesion causing Horner's syndrome was in this case near this ganglion?

INCOMPLETE HORNER'S SYNDROME

Occasionally a patient is seen in the clinic with an incomplete Horner syndrome. For some reason, difficult to explain, the only nerves involved are the postganglionic fibers to the smooth muscles of the upper lid, which results in drooping of that lid. It is practically impossible to distinguish this drooping from mild ptosis unless the instillation of epinephrine hydrochloride into the conjunctival sac is utilized. If the lid regains its normal position after the application of this drug, ptosis can be excluded. The instillation of epinephrine hydrochloride in such cases is not only of diagnostic but of therapeutic value. I knew a young girl with a drooped upper lid of one eye, who, on my advice, instilled a few drops of epinephrine hydrochloride into the conjunctival sac of the affected eye, whenever she went to a dance or to some social affair. The lid maintained its normal position for a few hours.

CASE 4.—A. B., a 30 year old woman, came to the clinic on Oct. 16, 1936, complaining that there had been something wrong with her eyes for the last three months. She had apparent exophthalmos of the right eye. The exophthalmometer readings were equal for the two eyes. This finding ruled out unilateral exophthalmos. What she actually had was drooping of the left upper eyelid, which gave that eye a less prominent appearance. The instillation of epinephrine hydrochloride into the conjunctival sac caused elevation of the drooped left upper lid. The patient then remarked: "That is the way my eyes were before the trouble started." The apparent exophthalmos temporarily disappeared.

CASE 5.—H. C., a girl 20 years of age, came to the clinic on Oct. 23, 1936, complaining of drooping of the right upper lid. The instillation of epinephrine hydrochloride did not cause elevation of that lid. Her trouble was retraction of the left upper lid. That this was the actual condition was shown by a photograph of the patient taken some time before her trouble started.

AURAL DISEASE AND IRRITABILITY OF THE DILATOR MUSCLE OF THE PUPIL

In the literature there are reported a number of observations of either a dilated or a constricted pupil in cases of lesions of the tympanic cavity.[5] Constriction of the pupil has been noted in chronic diseases of the middle ear in which destruction of the pupillary dilator fibers on the promontory occurred. In acute infections of the middle ear, dilatation of the pupil on the affected side may appear, due to an irritation of the aforementioned fibers. In practice these pupillary changes are rarely observed, but they can be made manifest by the instillation of epinephrine hydrochloride into the conjunctival sacs. An example is the following case of Dr. H. P. Davis.

CASE 6.—J. R., a 34 year old man, came to the clinic on Sept. 10, 1931. He complained of pain, discharge and deafness in the right ear. He had two operations on that ear: A simple mastoidectomy was done when he was 11 years old, and a radical mastoidectomy was done in 1925. A secondary radical mastoid-

ectomy was performed on Sept. 17, 1931. When he returned to the clinic on July 29, 1935, there was a mass that filled the right mastoid cavity, and on neurologic examination there was found paralysis of the sixth, seventh, eighth, ninth, tenth, eleventh and twelfth cranial nerves. The microscopic diagnosis of the mass was hemangio-endothelial sarcoma. The pupils were of normal size and were equal. However, the instillation of epinephrine hydrochloride into the conjunctival sacs caused dilatation of the right pupil but not of the left pupil.

GLAUCOMA AND PREGLAUCOMATOUS STATES

Simple primary glaucoma is accompanied by irritation of the sympathetic nervous system of the eye, which can be made manifest by dilatation of the pupil following the instillation of epinephrine hydrochloride into the conjunctival sac. I have observed a few patients who had glaucoma in one eye only, the other eye being normal as far as could be determined; yet both pupils dilated after the instillation of epinephrine hydrochloride. Arnold Knapp[6] reported his observations of the action of epinephrine on the pupil in sixty-five cases of primary glaucoma. In sixty of these cases the pupil became dilated after the instillation of epinephrine hydrochloride. In fifteen of these sixty-five cases the glaucoma was apparent in only one eye. The other (normal) eye showed in twelve cases dilatation of the pupil after the instillation of epinephrine hydrochloride. Dr. Knapp made the significant statement: "This suggests that a susceptibility to adrenalin may be present long before the clinical signs of glaucoma." One may then assume that hyperirritability of the sympathetic nervous system of the eye is an accompanying sign of a preglaucomatous state.

Every ophthalmologist has observed that, in preparing patients for operations on the eye, in those of late adult or advanced life, the pupil of the eye to be operated on may sometimes dilate after the instillation of epinephrine hydrochloride. If all other causes of irritability of the sympathetic nervous system of the eye are excluded, is one not justified, in the present state of knowledge, to consider these patients as being susceptible to glaucoma and to take the necessary precautions to prevent, if possible, this dreadful disease? This phenomenon does not necessarily mean that glaucoma will develop in these patients. It only means that they have irritability of the sympathetic nervous system of the eye, and this condition precedes and accompanies glaucoma.

SUMMARY

Epinephrine hydrochloride, in the strength of 1:1,000, when instilled into the conjunctival sac, will stimulate the sympathetic vasoconstrictor nerves of the eye, but not the sympathetic fibers to the dilator muscle

6. Knapp, Arnold: The Action of Adrenalin on the Glaucomatous Eye, Arch. Ophth. **50**:556, 1921.

of the pupil in a normal person. In order to produce mydriasis by epinephrine, the sympathetic nervous system of the eye must be in a state of hyperirritability. This hyperirritability can be brought about by: (1) exophthalmic goiter; (2) diabetes; (3) certain drugs, such as cocaine; (4) irritation of the sympathetic fibers in any part of their course from the superior cervical ganglion to the dilator muscle of the pupil, which may occur in acute disease of the middle ear, or other conditions, such as new growths that produce pressure; (5) destruction of the superior cervical ganglion and its postganglionic fibers to the dilator muscle of the pupil, resulting in denervation of this muscle, which may occur in chronic diseases of the middle ear and in some cases of Horner's syndrome; (6) glaucoma, and (7) preglaucomatous states.

CONCLUSIONS

In testing out the effect of epinephrine on the dilator muscle of the pupil of many hundreds of patients in the Manhattan Eye, Ear and Throat Hospital, during the last eighteen years, I have become convinced that there is always a definite reason whenever a pupil dilates on the instillation of a solution of this drug into the conjunctival sac. That reason is hyperirritability of the sympathetic nervous system of the eye. The underlying cause of this hyperirritability remains to be determined.

161 East Seventy-Ninth Street.

TREATMENT OF DENDRITIC KERATITIS WITH QUININE BISULFATE

ELIAS SELINGER, M.D.

CHICAGO

Dendritic keratitis seems to be a much more uncommon disease in the Middle West than in the New England states. Gundersen [1] reported on the treatment of two hundred and twenty-one patients with dendritic keratitis seen within forty-two months. He found the incidence to be highest before the tenth year of life and reported good results from cauterization of the cornea with strong solutions of iodine.

In the past two years eleven adults with simple dendritic keratitis—no children with this disease were seen—were treated with quinine bisulfate in addition to the usual treatment with hot compresses, atropine and bandages. An ophthalmic ointment containing 2 per cent quinine bisulfate was applied to the unanesthetized conjunctival sac twice a day by the patient. No massage was used. In none of the cases was there any extension of the process, as evidenced by the fluorescein stain. The subjective symptoms improved from the beginning—possibly as a result of the anesthetic action of quinine on mucous membranes—and in most of the cases the corneal lesions healed within from a week to ten days. In practically all the cases the eyeball was pale within one or two weeks. In no case did the disease last more than three weeks. In all the cases the condition was moderately severe, and in some there was considerable iritic irritation. It is possible that in instances in which the condition was unusually severe the course of the disease might have been more prolonged. No complications in the form of hypopyon keratitis were observed, and up to the present recurrences have not been seen. Most of the patients had an accompanying or preceding infection of the upper respiratory tract. In one case the onset followed removal of a foreign body from the cornea. None of the patients had malaria.

The good results obtained with local quinine therapy suggest a destructive action on the virus producing the disease. The same treatment was also tried in a case of a related virus disease, namely herpes zoster with corneal involvement. The result was good, the corneal lesion going on to healing within three weeks, but as herpes zoster corneae is

From the Department of Ophthalmology of Rush Medical College, the University of Chicago.

1. Gundersen, Trygve: Herpes Corneae, with Special Reference to Its Treatment with Strong Solution of Iodine, Arch. Ophth. **15**:225 (Feb.) 1936.

uncommon, no occasion to check the results has presented itself, and no conclusions can be drawn from the results in a single case.

In dendritic keratitis this therapy compares favorably with any other form of treatment previously suggested and because of its simplicity and the lack of discomfort accompanying it is preferable to more strenuous therapeutic measures. The burning sensation which follows the application of the quinine bisulfate ointment is comparatively slight, the drug having been used successfully in children from 2 to 5 years of age for other conditions, as was previously reported in other communications.[2]

This report is submitted in order to stimulate the investigation of the action of quinine in the treatment of dendritic keratitis in regions where this disease is more prevalent.

58 East Washington Street.

2. Selinger, Elias: Local Quinine Therapy in Cases of Interstitial Keratitis and Old Corneal Opacities, Arch. Ophth. **13**:829 (May) 1935; Local Quinine Therapy for Some Diseases of the Conjunctiva and Cornea, ibid. **15**:31 (Jan.) 1936.

Clinical Notes

QUICK TEST FOR PRESENCE OF A VERTICAL PRISM PRODUCED BY A DECENTERED LENS OR A CROOKED FRAME

LOUIS BOTHMAN, M.D., CHICAGO

For many years I have used a Maddox rod with a double loop of piano wire arranged as shown in figure 1 to hang over one lens of a pair of glasses to determine quickly whether a decentered lens or a

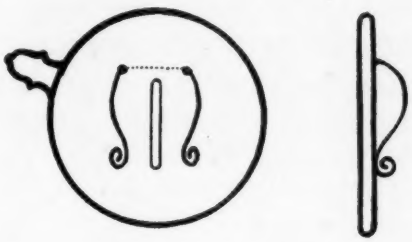

Fig. 1.—Modified Maddox rod to be hung over the lens.

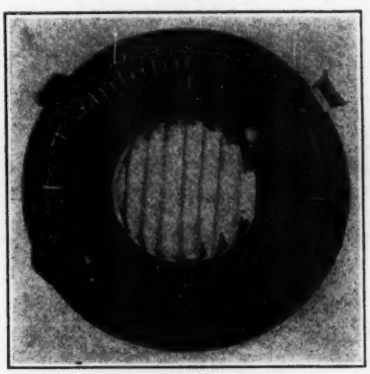

Fig. 2.—Metal disk with circular area of glass rods, to be hung over the lens.

crooked or bent frame is producing a vertical prism. I have been using a Maddox rod made of red glass and a round electric globe which I employ as my source of light in doing retinoscopy. The patient quickly and easily sees the red horizontal stripe and can tell whether it goes through the center of the light or not. The test is performed so quickly that it has become a routine procedure in checking lenses when the patient returns with his glasses. The same procedure can be carried out

From the Division of Ophthalmology, the University of Chicago, Dr. E. V. L. Brown, Director.

with the use of the Maddox rod in the horizontal position to check horizontal phoria, but this is not done as a routine.

I prefer an ordinary Maddox rod, because its light weight does not pull down the lens over which it hangs.

Figure 2 shows a metal disk with two hooks to hang over a lens, made for me by the American Optical Company. In this disk is a circular area of pieces of glass like those of a Maddox rod, which can be turned to produce either a vertical or a horizontal streak of light. This disk is too heavy and causes the lens to sag, so I have discarded it and use the rod shown in figure 1.

Of course, holding a rod in front of a lens will give the same results, but because this presses the lens out of position and must be raised or lowered or turned from side to side, I have found it more convenient and quicker to use the rod hung over the glasses.

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Ophthalmologic Review

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FITTING OF CONTACT LENSES FOR PERSONS WITH AMETROPIA

EVOLUTION AND MODERN TECHNIC

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NEW YORK

Because of recent developments which have added to the comfort of the patient and the widespread publicity given to this new device for correcting ametropia, contact lenses deserve the careful and thorough consideration of the ophthalmologist. He will be asked many times in the near future the uses to which these invisible lenses may be put, the comfort with which they may be worn, the chance of injury through breakage, how long they have been used, how they work, what chance there is for irritation, how they are inserted and removed and other questions which only the ingenuity of a patient aroused by curiosity could conceive.

Contact lenses, while originally designed to correct keratoconus, have been suggested and used for many conditions other than that for which they were principally intended.

They have been used as a protective covering for the cornea in cases of entropion, trichiasis, keratitis e lagophtalmo, neuroparalytic keratitis and keratocele, and to protect the eye from chemical fumes and burns.

As a refracting device they have been successfully employed in cases of keratoconus, corneal scars, protruding degeneration of the cornea, leukoma, descemetocele and ametropia. The classification ametropia includes myopia, hyperopia, aphakia and corneal astigmatism.

Occupationally they are valuable to ship officers and yachtsmen bothered by spray when ordinary spectacles are worn. Actors and actresses of the stage and film, clergymen and lecturers are using them because they hide their defective eyesight and give them the security of normal vision.

Automobile drivers use them because the disturbing and often dangerous reflections on spectacle lenses are avoided.

Swimmers use them because they will not come off during diving or swimming in rough water.

Hunters, sportsmen, athletes and aviators find them more practical than spectacles because of the increased field of vision, the lack of hindrance and the fact that the danger of breakage is practically nonexistent.

The cosmetic effect of these invisible lenses appeals to both young and mature women who ordinarily would have to wear heavy plus and minus corrections. Recently a large number of young women, mostly myopes, have requested contact lenses to correct ametropia as low as 2 diopters, so that they may enjoy good vision during social affairs without the use of spectacles and eyeglasses.

In special instances light-absorbing corneal portions are prescribed for albinos and as a protection from snow blindness. Pinhole lenses with translucent media are also available as an aid to vision.

The danger of breakage is practically nonexistent, as the contact lens lies in a protected position beneath the eyelids and therefore is afforded the natural protection given to the eye itself by the bony structure of the anterior part of the orbit. There is no case on record in which injury has resulted to an eye through breakage of a contact lens.

In one case on record the contact lenses served as a protection. The whole face was badly cut in an automobile accident; there was a gash across the forehead, cheek and eyelid, but the eye was protected by the contact lens. Zeiss contact lenses resist breakage even when dropped. In two instances patients have knocked contact lenses onto a tile hospital floor from a height of from 3 to 4 feet (90 to 120 cm.). The astonishing fact is that they merely bounced and settled uninjured and were ready, after sterilization, for further use. They are breakable, nevertheless, if they happen to land on the edge of the glass. One patient reported that a lens was broken from a drop into a wash basin, and during a trial fitting one lens was dropped on the metal foot rest of an operating chair and broke in three pieces. Needless to say, they will not survive being stepped on.

The comfort of the patient and the length of time the lenses may be worn depend on several factors. The accuracy of the fitting, the physical make-up and attitude of the patient and the ease with which tolerance is established are all concerned. It may be generally stated, however, that if the patient can successfully wear the trial lens for three or four hours, the chances of trouble with the finished lens are nil, and the patient can be assured that he will be able to wear it for from eight to twelve hours after tolerance has been established.

Contact lenses are superior to spectacle lenses because it is optically impossible to obtain equally good vision over the entire surface of a lens placed 12 mm. or more from the eye. This is especially true in the case of high refractive errors in which there is some distortion about the edges of the correcting lens, limiting the field of useful

vision. The contact lens, worn on the eyeball and moving with it, becomes a part of the refractive system of the eye and produces the same correction, regardless of the position of the eye. In myopia the retinal image is enlarged as compared to that seen with spectacle lenses, giving added visual acuity. In antimetropia and anisometropia binocular vision is possible with comfort. In aphakia, when only one eye has been operated on, binocular vision can be maintained when the aphakic eye is corrected with a contact lens. This is impossible with ordinary spectacle lenses, owing to the aniseikonia produced.

Further, contact lenses are not made useless by rain, snow, sleet, mist and steam, as are spectacle lenses.

Further detailed description and comment will follow as various phases of this work are discussed.

HISTORICAL BACKGROUND

The original idea of covering the cornea with a glass shell, from which the contact lens subsequently developed, originated over a hundred years ago. In 1827, J. F. Herschell, the well known English astronomer, suggested the use of such a device to protect the cornea from a diseased lid. The second record of such a device is the statement of F. E. Müller that in 1887 Saemisch sent to the Müller firm a patient suffering from keratitis e lagophtalmo due to a cancerous lid. This organization made a thin blown glass covering to protect the cornea from further ulceration. It was reported that the patient wore this protection for several years. No attempt or suggestion was made up to this time to correct errors of refraction by these devices.

For a considerable time investigators had been experimenting with means of restoring the visual acuity of persons with keratoconus. They were discouraged with the lack of improvement in vision obtained with spherical and cylindric lenses. The latter were not used at this time to any extent, and no accurate means of measuring the corneal irregularity was possible until the invention of the ophthalmometer in 1882.

About 1879 Rahlmann suggested the use of hyperbolic lenses for the correction of keratoconus. He was probably the first to try such lenses in a spectacle frame. Angelucci in 1884 also attempted to obtain an increase in vision with conical spectacle lenses in cases of the same type.

Both these attempts proved to be unpractical. Improved vision resulted as long as the optic axis of the corneal cone coincided with the optic axis of the conical lens. However, the slightest displacement of the two axes, as the eye rotated, caused considerable distortion, with loss of visual acuity. The visual field was therefore extremely limited and may be compared to that obtained through a pinhole placed from 10 to 20 mm. from the eye.

Contact lenses came into being in 1888 as a result of the efforts of A. Fick to restore the visual acuity of patients with keratoconus. To him belongs the honor of having been the first to suggest and use any glass shell covering the cornea to correct visual errors. He requested Ernst Abbe, associate of Carl Zeiss, to grind a contact glass for him that would have a corneal curvature of 8 mm. and a small scleral band with a curvature of 12 mm. The Zeiss firm sent him several blown lenses instead, with which he conducted his experiments.

The principle concerned with the use of these lenses is the neutralization of all the astigmatism produced by the keratoconus with a liquid lens of approximately the same refractive index as the cornea, formed between the anterior surface of the cornea and the posterior surface of the contact lens. In this way the refracting surface is moved from the highly astigmatic surface of the cornea to the inner spherical surface of the contact lens.

Fick continued his experiments by making plaster molds of cadavers' eyes over which further contact lenses were blown. In the same year (1888) August Müller constructed a contact lens which improved his own highly myopic vision.

Sulzer reported in 1892 that he had obtained good vision with ground contact glasses in one case of high astigmatism and in two cases of keratoconus. While these lenses were a slight improvement over those of Fick, they were not of practical value. When worn a short time they produced marked congestion of the eye and erosions of the cornea. This was due to the fact that they had very narrow scleral portions and were designed to rest on the outer edge of the cornea, where pressure at the limbus soon caused pain, lacrimation and severe photophobia due to the dilatation of the blood vessels. The lack of a wide scleral band made all contact lenses so far produced unwearable for any length of time, and the idea of contact lenses was abandoned for a number of years. However, a protective glass shell had progressed to an actual contact lens which was able to correct visual errors as long as it could be tolerated, even though that period was a short one.

Before the revival of an interest in contact lenses the principle behind them was used by Lohnstein, of Berlin, in 1896 and Siegrist, of Switzerland, in 1897 in a device called the hydrodiascope. This instrument consists of a small chamber which is filled with saline solution in contact with the eyeball through the eyelids, which are also covered by the saline solution. The anterior portion consists of a glass window in front of which varying spherical lenses can be placed for distance or for reading vision. All corneal irregularities are neutralized by the water lens formed in the same manner as with the contact lens. The hydrodiascope improves vision considerably and can be worn with comfort over long periods. The disadvantages are its disfiguring

appearance and the loss of field caused by the addition of high power spheres which have to be used to compensate for the normal corneal curvature of about 44 D., which is reduced to a much lower value by the glass front of the device. The hydrodiascope is still manufactured today by a Swiss organization.

MÜLLER GLASSES

The marked improvement in visual acuity that resulted from the work of Fick and Sulzer finally led to further experimentation with contact lenses.

The next development to appear was the blown type of contact glass manufactured by the Müller organization of Wiesbaden in 1909. F. A. Müller's contribution to the solving of the problem of contact lenses was the addition of a wide scleral flange which gave contact over a large portion of the sclera and enabled the corneal section to clear the cornea and limbus entirely.

On casual examination the Müller glass presents much the same appearance as an artificial eye, except that the iris is replaced by clear glass. It is much thinner in form, about 0.5 mm. in thickness and has a clear central corneal area surrounded by an opaque portion of irregular shape which resembles the normal sclera in color and in the design of blood vessel. It is therefore inconspicuous when worn. The scleral portion is from 7 to 8 mm. wide in the lower flange, from 4 to 5 mm. wide in the upper flange and from 10 to 12 mm. wide in the temporal and the nasal portion. There is a gradual transition from the corneal to the scleral part.

The corneal portion of the Müller glass cannot be made with a perfect spherical surface because it is blown. Siegrist reported in 1916 that of fifty-two Müller lenses examined, twenty-seven showed a slight astigmatism at oblique axes, and twelve had a high astigmatic error. In other words, 75 per cent of the lenses examined showed astigmatic defects. No grinding to produce a perfect scleral portion or to add refracting power is possible on the corneal portion of the Müller lenses. Erggelet and von Hippel sent a large number of Müller lenses to the Zeiss Works for grinding. Every glass broke in the process. Müller ascribed this to the thinness and delicacy of the shell rather than to quality of the glass. It is also probably due to internal stress in the glass itself, due to lack of proper annealing.

Therefore, because of corneal defects and lack of definite corneal curvatures, these lenses must be selected empirically by trying out a large number on the patient's eyes. Because both the scleral and the corneal curvature must be selected in the same manner and a good scleral or corneal fit is seldom combined in the same lens, this is an arduous task for both the physician and the patient. Trial with from

thirty to fifty glasses for each eye is not unusual in a number of cases. If the lenses are lost, scratched or broken the entire process must be repeated.

The literature on contact lenses up to and including 1931 reports experiences of greater comfort with Müller glasses than with Zeiss contact lenses. Lindner and Erggelet in 1930 and Sitchevska in 1931 brought out this point. These reports were undoubtedly true at the time they were made. One must take into consideration, however, the fact that the first Zeiss lenses used up to this time were made with only one scleral radius, of 12 mm., with which to fit all eyes.

Sitchevska suggested that the broad scleral part of the Müller glass, being supported by the lower fornix, forms a supporting base for the corneal portion. Pressure on the eyeball, if present, is comfortably distributed, as it is minimized in inverse proportion to the broad scleral band. Further, the gradual transition from the corneal to the scleral segment prevents pressure on the limbus and ciliary vessels; hence there is no interference with nutrition. The last point is not of much importance today, because the Zeiss lens is obtainable with a continuous and gradual transition from the corneal to the scleral portion.

Three patients, during the last six months, have reported discomfort and lack of vision with the Müller type of lenses. Two have tried from forty to fifty lenses, and the third complained because the broad scleral portion prevented the glass from moving freely with the rotation of the eyeball.

Clausen in 1920 reported that the ground lenses became roughened in grinding and therefore produced irritation and congestion. No such condition exists today, owing, no doubt, to advanced methods of polishing with wax and pitch.

Sommer in 1927 and Gill in 1928 reported the Müller glass as being more durable, and less irritating because of the smooth fire-polished surface and, further, as being chemically unaffected by the lacrimal fluid. Lauber reported on a patient who wore the same glass for six years. Contrary reports were made recently by two patients wishing to be fitted with Zeiss contact lenses. They had previously been wearing the Müller type of lenses. One had to change the glass every eight months because of roughening of the posterior surface of the glass by chemical reaction of the secretions of the eye, and the second could wear his for only twelve months for the same reason.

As the Müller type of glasses are blown from "glass eye" glass, it is reasonable to expect them to react in a similar manner as glass eyes. The life of a glass eye is generally known to be about from eight to eighteen months. At the end of this period the glass is so roughened and checked by reaction with chemicals of the body that it must be replaced.

This is understandable when consideration is given to the fact that the glass used has been developed for blowing quality and naturalness of appearance in the finished product. Wearing quality is a secondary consideration. Further, there is no difference in the quality of the glass used in glass eyes and that used in blown contact lenses, because absolutely all the glass is obtained by the various manufacturers from one firm in the small German town of Lauscha. One family has held and jealously guarded for generations the secret of the manufacture of this unusual glass.

During 1934 a New York firm started the manufacture of a Müller type of contact glass. It has an improved corneal portion on which correcting powers may be blown. While lenses of this type have proved satisfactory in some cases, three patients with high myopia have reported that useful vision has not been obtained with them, although a large number were tried. Two other patients reported that comfortable scleral portions were yet to be given them, although forty or more lenses had already been fitted. All five patients have requested a fitting of the Zeiss contact lenses.

The insertion and removal of the Müller lenses are comparatively simple and are learned by the average patient in one visit; they do not require a local anesthetic. The glass, with the concave surface up, is held between the thumb and the index finger. The glass is filled with half-normal sterile solution of sodium chloride at body temperature. The patient then looks down, raises the upper lid with his other hand and inserts the upper portion of the lens under the lid. The upper lid is then released and the lower lid pulled down as the patient raises his eyes, and the glass is inserted under the lower lid, which is in turn released.

If bubbles of air are formed under the glass they can be released by lifting the upper edge of the glass. If this fails, the glass must be removed and reinserted.

The removal of the glass is more difficult for some patients at first. The patient should look up, lift the lower edge of the glass with his finger-nail and push it out by contraction of the upper lid. A glass or ivory spatula or a dull flat muscle hook often helps to lift the lens away from the sclera. Patients have reported, however, that these devices often simply push the glass farther up under the upper lid. The placing of a finger over the upper eyelid at the upper edge of the contact glass will prevent this. It is also possible to remove the Müller type of lens by grasping it with the thumb and the index finger at the inner and outer canthi and, while pulling the lower lid down with the other hand, gently pull the glass down and out from the eye.

ZEISS CONTACT LENSES

Carl Zeiss accepted the suggestion of a wide scleral band, as advised by Müller, and finally produced ground contact lenses having this feature. They present the appearance of a thin saucer-shaped shell of colorless, chemically resistant special optic glass formed to correspond to a cross-section through the anterior two fifths of the globe. They consist of two sections of different radii so that the outer portion will rest on the sclera and the central portion will curve over the cornea and limbus without touching them (fig. 1). The edge dividing the two portions and the rim are carefully rounded and polished.

The corneal portion consists of an accurately ground spherical shell designed to correct defective vision. Because the diameter of the average cornea is approximately 11 mm., the diameter of the corneal section has a diameter of 12 mm., which is ample in most cases. The scleral rim is 4 mm. wide, making the entire diameter of the standard contact glass 20 mm. The scleral band can be ground narrower, wider or oval, if so desired for special cases.

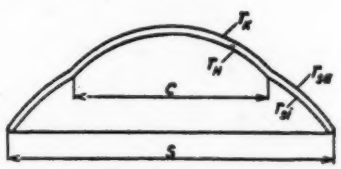

Fig. 1.—Ordinary trial contact glass 2/8, without refractive power, with a radius of curvature of the inner corneal surface (r_H) of 8 mm. and of the inner scleral surface (r_{sl}) of 12 mm. The diameter of the boundary edge of the corneal and scleral portions (c) is 12 mm. The over-all diameter (s) is 20 mm.

This type of contact glass was first used and demonstrated by Stock in 1920, causing a revival of an interest in the ground type of contact lenses. Unfortunately the first lenses of this type were made in only one scleral radius, of 12 mm., and in four corneal radii, of 6.5, 7.1, 8.1 and 9 mm., respectively. They were intended only for the correction of keratoconus, and the corneal curve giving the best vision was selected, added power being supplied by spectacle lenses worn in front of them. Stock, who had keratoconus himself, reported perfect vision with the new lenses but great discomfort after they had been worn for two or three hours.

It was soon apparent that although the eyeball has an average diameter of 24 mm., a constant radius of 12 mm. on the scleral portion proved unpractical in many instances. The next step in the development was the manufacture of contact lenses having scleral radii of 11, 12 and 13 mm., combined with varied corneal curves.

In 1929 Professor Heine, an ophthalmologist of Kiel, Germany, read a paper on his work with contact lenses before the Thirteenth Inter-

national Congress of Ophthalmology, at Amsterdam, Netherlands, which caused world-wide interest in their use. Heine suggested and demonstrated the practical application of contact lenses for correcting ametropia due to myopia, hyperopia and astigmatism. Thus contact lenses were brought to the aid of the average patient with the more usual forms of defective vision.

The principle of Heine's method consists of substituting for the anterior refracting surface of the cornea the posterior surface of the contact glass. The fluid lens formed between the cornea and the contact glass has approximately the same index of refraction as the cornea and practically eliminates the optical action of the latter. In this way any corneal astigmatism or any other refractive errors caused by an irregularity of the cornea are neutralized.

The power of the fluid lens is determined by the radius of curvature of the spherical posterior surface of the contact glass, at which surface its anterior curvature is formed.

In other words, the corneal refracting media, with the contact glass on the eye, consists of a lens formed by the posterior surface of the cornea and the posterior surface of the contact glass. The corneal substance and the fluid lens can be considered optically as a homogeneous whole.

By varying the radius of curvature of the corneal portion of the contact glass to a value different from that of the patient's cornea, the focal point of the entire system may be moved forward or backward according to whether hyperopia or myopia is to be corrected. Thus, a myopic eye having an axial refraction of —5 D. with a corneal radius of 8 mm. can be corrected by an afocal contact glass having a corneal segment with a radius of 9 mm. on the posterior surface.

This is a method of correcting ametropia equivalent to remolding the anterior surface of the cornea to suit one's needs.

The actual procedure of Heine's method of determining the proper contact glass to meet the needs of each patient is as follows:

The radius of curvature of the anterior surface of the cornea is measured by means of an ophthalmometer. When the cornea shows an astigmatic condition so that two ophthalmometer readings result, either of the two may be chosen for the purpose of computation.

The axial refraction of the eye is determined subjectively or objectively.

With this information at hand, let A equal the axial refraction in diopters; C, the ophthalmometer reading in diopters, and R, the radius in millimeters of the posterior surface of the corneal portion of the contact lens used to correct A.

The formula is $R = \frac{336}{A + C}$

The solution of the problem is simple as far as the needed corneal radius is concerned.

According to Professor Heine, after the axial refraction, A, and the radius of curvature of the cornea, rc, have been determined, the proper contact glass for complete or nearly complete correction may also be obtained with the aid of a double scale (fig. 2) giving the radii of the curvature, rc, together with the corresponding refractive powers of the corneal system, Dc. For example, let the axial refraction, A, equal —6 D, and let rc equal 8.1 mm., which, according to the double scale, equals 41 D. (dc). To this must be added the diopters to be corrected, or the axial refraction, A. Then 41 D. — 6 D. = 35 D. Under +35 D. on the double scale is the equivalent value rc = 9.5 mm. The myopic eye would see properly if the cornea had a radius of curva-

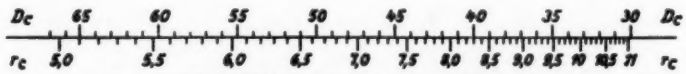

Fig. 2. Double scale with corneal curvature radii r_c and the corresponding refractive powers D_c in the corneal system. 13383

Fig. 2.—Double scale with radii of the corneal curvature (r_c) and the corresponding refractive powers (D_c) in the corneal system.

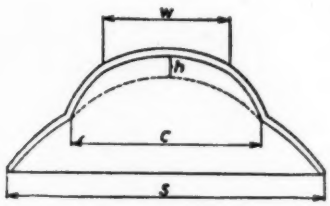

Fig. 3.—Contact glass with a raised corneal portion. Between the optically effective part of the cornea of the restricted diameter (w) which is 8 mm., and the scleral portion, with its usual diameter (c) of 12 mm., there is a ring zone. The corneal portion is raised by 1.3 mm. relatively to a corresponding contact glass of the ordinary type. The contact glass shown here has no refractive power; the radius of the inner corneal surface (r_H) is 8 mm., and that of the inner scleral surface (r_{s1}) is 12 mm. The over-all diameter (s) is 20 mm.

ture (rc) of 9.5 mm. Accordingly an afocal contact glass in which the anterior corneal radius equals 9.5 mm. must correct the error of refraction.

If A = 4 D. and rc = 7.9 mm., Dc would equal ±42 D. Then 42 D. + 4 D. = 46 D., and, again referring to the double scale under Dc 46 D., one finds 7.2 mm. The correcting contact glass would then have a radius of curvature of 7.2 or 7 mm. combined with an axial refraction of —1.5 D. or a radius of curvature of 7.5 mm. combined with an axial refraction of +1.75 D.

The correctness of the contact glass and the additional refractive powers obtained by the formula or the double scale must always be checked by subjective or objective methods of refraction.

When additional refractive powers over 4 D. are needed, compensation for the distance from the vertex of the trial contact lens to the trial spectacle lens must be calculated with the Zeiss variopter optical slide rule, or this distance may be stated when the lenses are ordered.

To correct high myopia according to Professor Heine's method, corneal radii exceeding 9 mm. are essential. With these the inner curve of the corneal part often rests on the corneal surface, causing irritation. In order to maintain the curves for the correction of myopia, the whole corneal part is raised 0.5 or 1 mm., as needed (fig. 3).

In 1930 the Zeiss Works announced Heine's suggestions and offered a trial set of contact lenses which comprised thirty-nine lenses

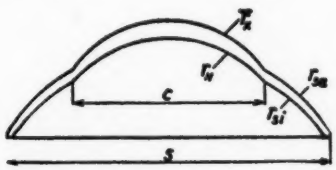

Fig. 4.—Contact glass 2/8+ 10 D. The center part is too heavy.

Fig. 5.—Contact glass 2/8 — 10 D. for permanent wear, with a radius of curvature of the inner corneal surface (r_H) of 8 mm. and of the inner scleral surface (r_{si}) of 12 mm. The glass has a refractive power of — 10 D. The divergent corneal portion, the diameter of which (c) is 12 mm., is considerably thickened at the edge. The over-all diameter (s) is 20 mm. The increased weight of this permanent contact glass, with its high corrective effect, sometimes proves a disadvantage in wearing.

having three scleral curvatures, of 11, 12 and 13 mm., and thirteen corneal curvatures, of from 5 to 10.5 mm., in differences of 0.5 mm.; it further stated that lenses having scleral curves with radii of 10.5, 11.5 and 12.5 mm. might also be obtained on special order.

These lenses were available with added refractive powers varying from +6.00 to —6.00 D. Soon higher powers were called for, but the lenses when so ground became too heavy because of the thickening in the center or at the limbus and proved unpractical (figs. 4 and 5).

The next two improvements in the Zeiss contact glasses appeared in 1932. First, lenses were made available with scleral radii in intervals

of 0.25 mm. and with corneal radii in intervals of 0.1 mm., thus adding a large number of lenses to those already available. Second, to take care of cases in which the corneal diameter exceeds the normal value from 11 to 11.5 mm. and in which the internal junction of the scleral and the corneal portion would therefore rest on or too near the limbus, contact lenses were ground with a continuous transition from the corneal curve to the scleral curve to avoid pressure at this point (fig. 6). This added to the comfort of a large number of patients who would otherwise be unable to wear contact lenses.

In 1933 J. Dallos, a Hungarian ophthalmologist, solved the problem of adding high corrective powers to any desired contact lens. He sug-

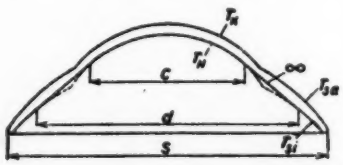

Fig. 6.—Contact glass with continuous transition from the corneal to the scleral portion of the inner surface. A conical surface touches the corneal portion in the circle of diameter c and the scleral portion in the circle of diameter d. The contact glass here shown has no refractive power; the radius of the inner corneal surface (r_H) is 7 mm., and the radius of the inner scleral surface (r_{si}) is 13 mm. The over-all diameter (s) is 20 mm.

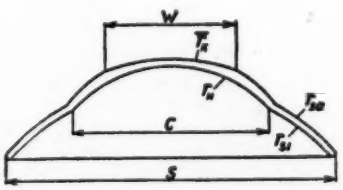

Fig. 7.—Dallos' permanent contact glass 3/8 —20 D., with a radius of the inner corneal surface (r_H) of 8 mm. and of the inner scleral surface (r_{si}) of 13 mm. Within the optically effective region of the corneal portion, of a diameter (w) of 8 mm., the glass has a refractive power of —20 D.

gested that the active corrective power be restricted to the central area of the corneal section within a diameter of 8 mm. Because the pupil is rarely as large as 8 mm. and because the contact glass moves with the eyeball, this area is ample. This method permits the addition of corrective spherical powers from —20 (fig. 7) to +18 D. (fig. 8) sphere.

Heine's method of fitting contact lenses has one decided disadvantage: It requires the investment of from $1,000 to $3,500 in trial lenses. This fact alone would prevent many ophthalmologists from working with these interesting and practical devices.

Experience has taught that this large number of trial lenses is unnecessary to do a good job of prescribing contact lenses, if one forgets the work of Heine and reconsiders the problem from another angle.

Ophthalmologists have at their disposal at the present time contact lenses having a scleral curvature with a radius of from 11 to 13.5 mm. in steps of 0.25 mm. These scleral curves are adequate to match accurately the scleral curvatures of the patient's eyes. This is the most important factor, for without a good scleral fit the lenses cannot be worn.

To meet the need of Heine's method of fitting contact lenses a vast variety of lenses having a corneal curvature with a radius of from 5 to 10.5 mm. in steps of 0.1 mm. are also available. However the number of corneal curves necessary to fit contact lenses can be reduced to three, those with a radius of 7, 7.5 and 8 mm., when all one has to consider is the clearance of the cornea, forgetting changes of refraction.

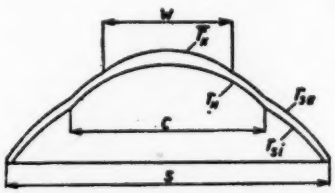

Fig. 8.—Dallos' contact glass 2/8 + 18 D. for permanent wear, with a radius of the corneal inner surface (r_H) of 8 mm. and of the scleral inner surface (r_{si}) of 12 mm. Within the optically effective region of the corneal portion, of 8 mm. diameter (w), the glass has a refractive power of + 18 D. The boundary edge between the corneal and the scleral portion has the usual diameter (c) of 12 mm., and the over-all diameter (s) of the glass is, as a rule, 20 mm. Notwithstanding its highly convergent effect, the corneal portion is only slightly thickened in the center. The weight of this glass is only a little greater than that of a contact glass without optical effect. The inner surface is identical with that of the corresponding trial contact lens 2/8.

Because of the work of Dallos, powers of from —20 to +18 D. can be ground on the anterior surface of the contact glass. One can add these powers to the front of the contact lens in the same way one prescribes spectacle lenses to fit in front of the cornea.

Therefore, the problem is reduced to fitting the patient physically with a comfortable contact lens which clears the cornea and fits well on the sclera, determine the refraction of the patient with this contact lens on the eye and prescribe a lens of the same size as the trial contact lens used plus the refraction required. The finished lens will be identical with the trial lens as far as the posterior scleral and corneal curvatures are concerned but will have ground on the anterior surface of the corneal portion the required spherical power.

A set of contact glasses containing lenses with curves having the radii listed in table 1 should prove adequate to fit the eye physically.

The corneal curve with the radius of 7 mm. is useful in cases of hyperopia; the corneal curve with the radius of 7.5 mm. is used mostly in cases of low myopia and in cases of hyperopic errors, and the corneal section with the radius of 8 mm. is used in cases of myopia. Of course these corneal portions can be used interchangeably on the same eyes if the refraction is changed.

FITTING CONTACT LENSES

The fitting of contact lenses, while comparatively simple, presents a number of interesting problems which call for considerable patience and mutual cooperation on the part of both the physician and the patient. Some persons are fitted with ease in three visits, and others require from six to ten visits before a satisfactory fitting is accomplished. The patient should clearly understand this and be willing to be guided by

TABLE 1.—*Set of Contact Lenses Adequate to Fix the Eye Physically*

Radius of Corneal Curve, Mm.	Radius of Scleral Curve, Mm.
6.5	12.50
7.0	11.5 11.75; 12.00; 12.25; 12.50; 12.75; 13.00
7.5	11.25; 11.5; 11.75; 12.00; 12.25; 12.50; 12.75; 13.00; 13.25
8.0	11.5; 11.75; 12.00; 12.25; 12.50; 12.75; 13.00

the physician's judgment and experimentation in overcoming the difficulties which his particular case involves.

Careful measurements of the corneal curvature should be made with the ophthalmometer and the radius of curvature in millimeters recorded. In cases of astigmatism in which two readings result, the corneal curvature should be considered as that of the shortest radius. If the ophthalmometer scale gives readings in diopters only, the diopters may be translated into millimeters of the radius by using the formula $M = \frac{336}{D}$, in which M equals the millimeters of the radius and D the diopters of corneal curvature.

The information thus gained will serve as a guide in choosing the corneal curvature of the contact glass first selected. In cases of myopia slightly longer radii may be tried, and in cases of hyperopia slightly shorter radii of curvature can be experimented with.

The scleral curvature is, at the present time, difficult to measure with instruments, and its determination is a matter of judgment and experiment. Several attempts to devise instruments for taking this measurement have been made. None has been entirely satisfactory. A new method, which will be referred to later in this paper, has solved the problem satisfactorily.

Before or after the ophthalmometer reading is taken 2 drops of a 1 or 2 per cent solution of phenacaine hydrochloride, butyn or some other local anesthetic which will not affect the accommodation or act as a mydriatic should be placed in the eyes to prevent reflex closing of the lids and all unnecessary distress when the various trial lenses are inserted and removed. If the trial continues over twenty minutes, additional drops should be given at intervals of twenty minutes. One of the principal difficulties still existing today is the lack of an atonic local anesthetic for use in this work. Often the anesthetic causes considerable irritation and dilatation of the conjunctival blood vessels and resulting photophobia. More often than not the irritation caused by the local anesthetic is greater than that caused by the contact glass when no anesthetic is used. An isotonic solution may help to reduce the irritation.

Fig. 9.—The contact glass on the rubber sucker, ready for insertion beneath the lids.

While the drops are taking effect the patient should be shown the contact lens, and assurance should be given that the process of insertion and removal will be painless and more simple than his probable mental picture of the procedure. A discussion of the ease with which some former patient inserts and removes the lenses will do much to quiet the patient's mind and will make it somewhat easier to work with him. Careful instructions as to just what is expected of him should be given at this time.

A small table should be prepared with all the necessary apparatus needed for the work to be done. This consists of the set of contact lenses, the sucker for inserting and removing the lens (fig. 9), gauze pads, absorbent pads, half-normal solution of sodium chloride or Ringer's solution, at body temperature, fluorescein, a good loupe, a small pencil electric flash-light, and a towel to protect the patient's clothes from any solution which may spill during the procedure.

A contact lens, sterilized by immersion in a 70 per cent solution of alcohol, is selected and cleaned with saline solution to remove all particles of dust. Some of the saline solution should be poured over the end of the rubber sucker to make it hold on the lens better. The lens is placed on the sucker, and only enough suction is used to hold it firmly. It is a mistake to get too much suction, because this makes it harder to release when the lens is in place. Often the contact lens will be pulled slightly away from the eye when the sucker is removed, and air enters back of the lens, causing a bubble, which often fills the entire corneal portion. The contact lens is not centered on the sucker but is placed at an angle, so that the shortest extending portion is toward the lower lid. When the lens is in place on the sucker it should be filled with half-normal solution of sodium chloride at body temperature.

The patient is seated close to the table and instructed to bend his head over with the chin well in toward the chest, in order to bring the eyes as near parallel to the top of the table as possible. He is asked to hold his lower lid down with his fingers over the eyelashes and instructed to release the lid slowly when requested. The person inserting the contact lens lifts the upper lid, keeping the lashes under the finger. When the lids are wide apart the contact lens is moved toward the eye, contact being first made near the lower lid, and then the lens is pushed close to the sclera under the upper lid. The upper lid is slowly lowered, the patient is instructed to release the lower lid, and the sucker is compressed to release the suction and then is withdrawn.

A second method of inserting the contact lens and the one the patient should be taught for his own use consists of filling the contact lens with saline solution at body temperature, raising the upper lid only and placing the contact lens under it and *over* the lower lid. The lens is held firmly in this position, with slight pressure toward the eye, and the lower lid is pulled down slowly until the contact lens jumps over the margin of the lower lid and falls easily into place on the sclera. This process is not advised if the lashes are very long. The patient can learn to do this easily himself. At first he should have a mirror placed almost flat on the table in which he can watch himself insert the lens.

Robert von der Heydt suggested that the patient stand on a carpeted floor, to prevent breakage if the contact glass should fall, and bend his head downward until his eyes are directed to his toes. With his arm over his head and using his most convenient finger, he should lift the upper lid away from the eyeball. The physician holding the contact lens filled with warm saline solution directs the edge of the contact glass under the margin of the upper lid and instructs the patient to release the lid, then draws the lower lid over the edge of the contact

glass and releases the latter by squeezing the nipple. If the patient cooperates and continues to look down at his toes, a bubble-free insertion will be accomplished.

In difficult cases in which the patient is very nervous and in which it is impossible to insert the contact lens without getting bubbles, the contact lens can be inserted dry with the patient's head in any comfortable position, and when the lens is in place on the sclera it can be pulled outward at the top with a dull horn spatula and filled with saline solution at body temperature with an eye-dropper. The two former methods are more satisfactory and should be used whenever possible.

With the contact lens in place the first check should be on the fit of the scleral portion. If a white or bluish white ring appears about

Fig. 10.—Diagram showing the pattern of the fit of the contact lens as revealed by fluorescein.

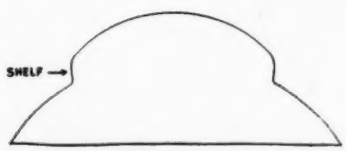

Fig. 11.—Shelf of the contact lens with greatly exaggerated raised corneal segment.

the edge of the scleral portion of the contact lens, the scleral portion is much too tight, and another lens with a longer scleral radius should be tried at once. Any disturbance in the flow of blood through the fine blood vessels of the surface is also an indication of the same trouble.

To remove the contact lens the patient can sit with the head in a normal position and the lids separated as when the lens is inserted. The sucker is placed on the corneal section of the contact lens and suction caused by squeezing and releasing the pressure. Too much suction cannot be produced at this time. The lens is pulled outward and downward. If it does not come off easily, a rocking motion from side to side may be given as the lens is pulled forward; as soon as the least bit of air enters back of the lens it comes off with no further trouble. At

times contact lenses are hard to remove, but this is due to improper fit. If they cannot be pulled down with the rubber sucker, a dull horn spatula should be inserted under the edge toward the outer canthus and the lens lifted until some air enters under the lens. The lens can be removed easily with the rubber sucker after this takes place. The contact lens which fits properly can be removed with comparative ease.

As soon as a scleral portion is found which does not cause a white line and does not disturb the blood vessels, several drops of fluorescein should be put in the eye. If this is dropped in at the upper edge of the contact lens, it will be likely to flow under it easier and will cause a greenish yellow color to appear under the contact lens wherever it does not touch the sclera or cornea. By the depth of color around the scleral portion it is possible to see how much of the scleral band is resting on the eye and how evenly it rests.

A valuable record of each scleral fit can be made by drawing the pattern seen with fluorescein for each trial lens used (fig. 10). This can be done rapidly. The darkened portion should represent the portion of the lens not in contact with the sclera. With this record at hand the same lens need never be tried a second time until the lens is selected for a final test of tolerance.

If the portion of the scleral band does not rest over too narrow a portion of the sclera, it is a good idea to use the slit lamp at this stage to check the fit of the corneal portion of the contact lens and to determine the distance between the cornea and the posterior surface of the corneal portion across its entire area. This distance can be estimated by the width of the quadrilateral image indicating the clearance as compared to the image which is caused by the thickness of the cornea itself, which averages 0.5 mm. in thickness. The clearance at the limbus should also be carefully checked.

If there is a good clearance between the lens and the cornea, the next larger scleral radius with the same radius of corneal curvature should be tried and the fit of the corneal portion checked again with the slit lamp. The purpose is to determine the contact lens which will fit over the largest portion of the sclera, clear the cornea and at the same time not rest on the sclerocorneal junction or on the limbus. It is possible for the contact lens to clear the cornea and fit the sclera too loosely, so that it will rest on the limbus and cause discomfort when worn a short time.

Caution should be used in prescribing raised corneal portions. They should not be ordered unless absolutely necessary, as in cases of keratoconus. The reason for this is the fact that a shelf is produced between the corneal portion and the scleral portion (fig. 11) which is struck by the movements of the edge of the lids, and the sclerocorneal junction

is forced against the limbus, causing pain, injection of the ciliary body and photophobia. The better procedure is to prescribe a shorter corneal radius to clear the cornea with added refractive power to compensate for the change in refraction. The vertex depth of the corneal portions, with a diameter of 12 mm., are approximately as shown in table 2.

Therefore, with the same radius of scleral curvature, there would be an additional clearance of 0.7 mm. when a radius of corneal curvature of 7 mm. is substituted for a radius of 8 mm., and when the radius of corneal curvature is reduced from 8 to 7.5 mm. there is an additional clearance of 0.3 mm.

In powers up to 9.75 D. it is possible to obtain contact lenses with a continuous transition between the corneal and the scleral curvature, thus doing away with any chance of pressure at the limbus. These lenses are ordered in the majority of cases in the lower powers. They are not advised in powers of 10 D. and above, because these lenses are ground, in accordance with Dallos' suggestion, with an active

TABLE 2.—*Radius of Corneal Curvature and Corresponding Clearance of Contact Lens*

Radius of Corneal Curvature, Mm.	Clearance, Mm.
9.0	2.30
8.5	2.50
8.0	2.75
7.5	3.05
7.0	3.45
6.5	4.00

refracting portion of only 8 mm. in the center of the corneal portion. The lenses with a continuous transition between the corneal and the scleral curvature fit too loosely for this type of correction and will allow the lens to slip too much to one side or the other and throw the refracting portion out of line. With lenses of lower powers this does not have to be taken into consideration, because the entire corneal portion is ground to the required power.

A great deal of time can be saved the physician and the patient if a proper mold is made of the anterior fifth of the globe and the contact lenses are fitted over the positive casting obtained in this way. It is seldom necessary to try more than one or two trial lenses if a careful preliminary fitting has been made over the castings. It should be possible to fit contact lenses in three visits if this method is used—one visit for the casting, one for the fitting and a third to determine whether a comfortable fit has been obtained. A set fee can be charged for the entire work and much valuable time saved. Detailed instructions for making the molds are given in the section dealing with Zeiss molded contact lenses.

As soon as a good physical fit is obtained the patient is instructed to return the next day or later. At this time the contact lens is inserted without an anesthetic, and the patient is told to wear the lens for two hours or until irritation shows before this time has elapsed. The troubles which appear at this time are tearing, inflammation and discomfort. If tearing occurs it is most likely due to the corneal portion's touching the cornea or the limbus. Inflammation and discomfort can be caused by too tight a scleral fit, by supersensitiveness of the conjunctiva, as in the case of sufferers from hay fever or merely by the contact lens' acting as a foreign body. If the last-mentioned cause is the difficulty and the congestion produced is not too great, satisfactory adjustment is merely a matter of establishing tolerance. The physician should determine whether this is the situation.

Indications which should lead the physician to advise against the use of contact lenses are: lack of comfort with the lens in place, a sensitive conjunctiva in patients with hay fever or other allergic conditions, tight eyelids which make insertion and removal of the contact lens difficult, vernal conjunctivitis, granular conjunctivitis pinguecula and abnormal conditions of the conjunctiva other than those mentioned.

It has been observed that during the trials or the two hour test period slight transient corneal opacities appear in some patients. The cause has not been definitely determined. It is possible that they are the result of osmotic changes due to the fact that the specific gravity of the solution of sodium chloride or Ringer's solution is too low or too high. An imbalance due to an improper p_H, or hydrogen ion concentration, has been suggested. Congestion caused by local disturbance of the blood supply has also been held accountable, particularly if pressure is exerted on the limbus. The physiologic make-up of the patient is most likely the cause of the trouble. Solutions of different strengths must be experimented with. Ringer's solution with a 0.1 to 1 per cent content of sodium chloride has been advised. The best average seems to be a content of sodium chloride between 0.7 and 0.8 per cent.

The determination of the p_H of the lacrimal fluid can be rapidly and simply accomplished with a new product, nitrazine paper. This resembles strips of litmus paper in size and texture and is impregnated with a sensitive indicator which changes from deep yellow (p_H 4) through mustard yellow (p_H 5), light olive green (p_H 5.5), olive green (p_H 6), blue-green (p_H 6.5), light olive green (p_H 7), blue (p_H 7.5) to deep blue (p_H 8) in easily distinguishable changes so that steps of 0.25 of a p_H interval can be accurately estimated when the test paper wet with a small amount of tears is compared with a chart provided showing changes in color. The normal moisture on the lids is sufficient in most cases. Full directions are supplied with the indicator paper.

Thirty persons were tested with this paper at random, with the results shown in table 3.

Whatever the cause of the corneal opacities, they seem to disappear a short time after the contact lens is removed. A drop of epinephrine hydrochloride used to reduce any slight irritation caused by the inserting of several lenses seems to make them disappear at once.

Congestion of the conjunctiva showing as conjunctival injection is often due to an imbalance caused by the lack of an isotonic balance between the fluid lens and the conjunctiva. Various strengths of sodium chloride and Ringer's solution as already advised should be tried to overcome this difficulty. Injection of the ciliary body with its accompanying photophobia is practically always due to pressure at the limbus.

A condition which is characterized by blurring of vision during the wearing and after the removal of a contact glass can also be attributed to a physiologic imbalance between the water lens and the conjunctiva, which causes an altering and thickening of the secretions which adhere

TABLE 3.—*p_H of the Lacrimal Fluid of Thirty Persons Tested with Nitrazine Paper*

p_H	Number of Subjects
7.3	5
7.5	12
7.6	6
7.7	8

to the cornea. This can be overcome or lessened by determining the proper type and strength of the solution for the fluid lens.

When, in the opinion of the ophthalmologist, the patient is properly fitted with a contact lens, careful refraction should be done. This will consist of the use of spherical power alone, as all the astigmatic error is neutralized by the water lens formed between the cornea and the corneal portion of the contact lens. The posterior surface of the contact lens is now the real refracting surface, instead of the anterior surface of the cornea. Any slight astigmatism apparent in the refraction is lenticular and cannot be corrected and for this reason must be ignored.

An interesting condition was observed in a patient of Dr. Walter Hipp. Before a trial of contact lenses this patient showed decided myopia combined with an astigmatic error of +0.25 D. in each eye. With contact lenses before the eyes, objective and subjective refraction showed lenticular astigmatism of +1.25 D. in each eye. In this case lenticular astigmatism neutralized an almost equal and opposite astigmatism of the cornea. It will be interesting to observe whether there is a change in the lenticular astigmatism when the corneal astigmatism is neutralized by the contact glass and the glass is worn for some time.

If the error of refraction is over 5 D., plus or minus, careful measurement of the vertex distance of the refracting lens to the anterior surface of the contact lens must be made and given to the Zeiss company when the lenses are ordered from them. This is very important in lenses of high power. The refracting portion of the contact lens is so close to the cornea that often a change of several diopters has to be made in order to give the same effective refraction as that obtained with the refracting lenses in the trial frame. This change in refraction can be calculated with the Zeiss variopter slide rule (Obrig 1935) and the actual power to be ground on the lenses may then be given, with no mention of vertex distance.

When the refractive error is low, the contact lenses should always be used in pairs. When the refractive error is above 5 D. it is often possible to use one lens. After a few weeks' trial, if discomfort is complained of the other lens can always be ordered. In high power, contact lenses can be successfully worn in one eye only, as the image from the unaided eye is easily suppressed.

The patient should be instructed to keep the contact glass clean. It should be sterilized every day with a 70 per cent solution of alcohol. Care should be taken to wash the alcohol off the lens with saline solution before it is inserted. It is important to be sure that no particles of dust are under the contact lens when it is worn. The lens can be filled with the saline solution and emptied several times before insertion in order to make sure it is clean.

The question is often asked how long contact lenses can be worn with comfort. It is advisable to be conservative in answering this question. Most patients can wear the lens for two hours twice a day as soon as they receive it. Tolerance must be established, and the patient should be instructed not to use the lens longer than two hours at a time during the first week. In most cases, by the end of the second week the lenses can be worn from five to eight hours a day.

One patient inserted a contact lens at noon on Sunday, lost the sucker for removing it and wore the lens until 10 o'clock the following Monday, at which time a new sucker was provided and the lens removed. In a case like this the lens could have been removed by lifting the scleral band with the finger-nail or a bent hairpin. However, as the patient was not uncomfortable she left the lens on for a full twenty-two hours. Absolutely no irritation was apparent when it was removed. Another patient wore a contact lens for thirty-six hours, with no irritation. The average period of comfortable wear is between eight and ten hours a day, after tolerance has been established. In all cases the eye must be educated to the fact that the contact lens is not a harmful foreign body. Several patients have reported moderate discomfort for

the first two hours, after which time no discomfort was felt, even though the contact lens was worn for ten or more hours. The first short period of slight irritation lasted in these cases only until tolerance was established.

MOLDED CONTACT LENSES

There are many patients who cannot wear the trial contact lenses with comfort for more than one-half hour. Ruling out those with a supersensitive conjunctiva and other contraindications to the use of contact lenses previously mentioned, this group consists of persons with gross irregularities of the sclera. The degree or irregularity varies from just enough to cause discomfort to as much as 1 mm. difference in the radius between axes at 90 degrees to one another. The large differences in curvature have been observed to consist mostly of short vertical radii and long horizontal radii of curvature. In the cases in which slight irregularities exist the usual condition is tight fitting at the approximate insertion of the internal and inferior rectus muscles, particularly, the internal rectus muscle, which is attached 5.5 mm. from the limbus, according to Fuchs.

Large differences in curvature are easily observed by dropping or lateral displacement of the corneal section of the contact lens in relation to the cornea. Slight differences in curvature are detected by pressure against the limbus at a point opposite the irregularity or by a difference in the depth of color of the fluorescein as previously described.

Regardless of the cause of the irregularity, patients showing such defects cannot be comfortably fitted with spherical contact lenses and must be informed that the only chance for comfort consists in having a cast made of the eyes and molded Zeiss contact lenses made from the casts.

Unfortunately, this process is experimental to some extent at the present time, and the patient should be informed of this fact. The only promises which can be made are that the physician and the Zeiss company will cooperate with the patient to the best of their ability and will endeavor to produce a comfortable contact lens. With proper cooperation between the three parties concerned the experimental phase will soon evolve into an extremely satisfactory method of fitting all contact lenses. At the present time, however, it will be more satisfactory to fit spherical contact lenses, whenever this is possible.

J. Dallos, of Budapest, Hungary, has reported the successful fitting of a number of molded contact glasses. He has called attention to the fact that the actual casting of the eye is the simplest part of the procedure. If the contact lens were blown over the cast as it is made, that is, if an exact replica of the anterior fifth of the patient's globe were made, the result would be a lens impossible to wear. The reasons

for this are the roughness of the conjunctiva and the fact that such lenses would rest directly on the limbus and the cornea.

In order to make molded contact lenses comfortable to wear from casts of the human eye, the casts themselves had to be modified before the lenses were blown against them. This gave rise to a system of haptics, a new branch of prosthetics. The original work was done in Hungary with the assistance of the Széchenyi Scientific Society. Thousands of experiments were made in which the shape of the casts was systematically changed before the lenses were blown against them. According to Dallos, "Each glass is made from a mold and according to the rules of haptics can be checked on the eye itself as to its size, shape, depth and curvatures, and if necessary its form can be corrected. After a perfect shell has been obtained the optimum dioptric effect is given by grinding the outer surface of the center which falls in front of the pupil" (fig. 12).

Fig. 12.—Molded contact lenses with a ground corneal portion.

Unfortunately, no report has been made of the rules of haptics, and ophthalmologists in America must depend on the Zeiss company to take care of that part of the work at present and confine themselves to furnishing the molds.

Making the Mold.—In order to obtain useful casts for fitting and molding contact lenses it is necessary to obtain a good impression of the cornea and a section of the sclera at least 5 or 6 mm. outward from the limbus. This has proved possible with the following procedure[1]:

The patient is placed flat on his back on a table or couch, with a small pillow for comfort, and instructed to gaze steadily at an easily

1. While the procedure described is somewhat different from that used by C. L. Stevens (A Method of Making Casts of the Human Cornea, Am. J. Ophth. **19**:593 [July] 1936) at the Institute of Ophthalmology of the College of Physicians and Surgeons and the Presbyterian Hospital of New York, her detailed description of her equipment and technic is of aid in making a large number of casts of the cornea in a study of keratoconus.

visible black marker on the ceiling. This should not be too small, for obvious reasons. Two drops of a local anesthetic is used to produce conjunctival anesthesia which will make the entire procedure painless and no more disagreeable than the taking of dental casts.

Three teaspoonfuls of Poller's negocoll is mixed with 1 ounce (30 cc.) of warm water in a double boiler in which the water is boiling in the lower section, or in a 50 cc. beaker immersed in an actively boiling sterilizer. The negocoll is stirred and worked with a palette knife or spatula into a paste free from all lumps, about the consistency of warm petrolatum or cold cream, warm water or additional negocoll being added if necessary.

When a homogeneous mixture is obtained, it is transferred to a 50 cc. beaker, if the mixing was done in a double boiler, and the small beaker is immersed in a 250 cc. beaker of cool water. The negocoll

Fig. 13.—Müller lens for casting and plaster cast.

is stirred with a glass stirring rod thermometer such as is used for photographic work. A second beaker of cold water should be at hand for use when the first cooling bath has become warm. The negocoll is cooled until the temperature is reduced to 102 F. At this temperature about ⅛ inch (0.3 cm.) of the negocoll paste is applied to the inside surface of a Müller type of contact lens, and the negocoll and shell are inserted under the eyelids as in fitting Müller lenses, care being taken not to exert pressure on the eyeball and the corneal section being approximately centered. No speculum is necessary and, in fact, is contraindicated when Müller lenses are used for making casts.

The Müller lens filled with negocoll is left on the eye with the lids in their natural position for two minutes. Then cold water is poured over it from an undine, the eyelids being separated somewhat at the same time, to set the gel. Absorbent material is placed to the side of the patient's head to absorb the waste water. The cast cools rapidly. The lower lid is retracted, and the mold is removed at once with a

muscle hook or a small horn spatula, which is carefully inserted under the lower edge of the Müller lens. The lens and the film of negocoll are carefully raised and drawn from under the upper lid, care being taken not to let the negocoll come in contact with the eyeball again (fig. 13).

Immediately on removal of the casting, the position of the inner canthus is marked with a notch, a red pencil mark or some other mark and is dropped, with the negocoll facing up, in a small beaker of cold water. The beaker is marked at once, O.D. or O.S., as the case may be, with a glass marking pencil. It is often confusing to identify the castings after they have been removed if they are not marked.

Dr. Harry Eggers suggested a modification of this process which has proved practical. In place of the Müller contact lens a special molding shell is used (fig. 14). This shell is 22 mm. in diameter and 7 mm. deep and has a glass handle 25 mm. long. The hollow blown handle

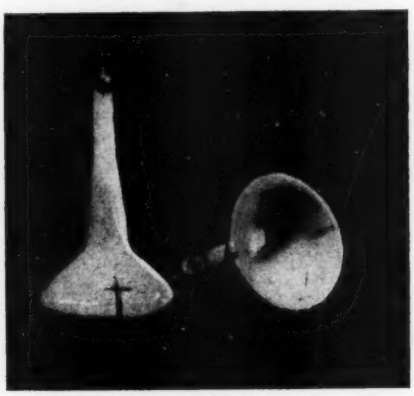

Fig. 14.—Casting shells.

should be plugged with cotton. The outside of the shell has a red dot or line for the right eye and a light blue dot or line for the left eye, welded near the edge to mark the position of either canthus.

The molding shell and negocoll are placed on the eye for two minutes, cooled with cold water and gently pulled away with the aid of the attached handle. Care should be taken not to fill the shell with too much negocoll, because the surplus runs out of the shell and jells under the upper lid, causing the film of negocoll to adhere to the eye instead of coming away with the molding shell when it is withdrawn. This film of negocoll is easily removed, however, and any loose portions can be washed from under the lid. The shells are then placed in a cold water bath supported by a small weighted wooden rack similar to a test tube holder, with the negocoll facing upward. The plaster casts can then be poured, with the molding shells in the same position on the rack (fig. 15).

Making the Plaster Cast.—After the molds have stood in cold water for thirty minutes, the surface is ready for making a cast (fig. 12). A good mold will show the impression of the cornea as a depressed shining surface surrounded by a more dull and slightly roughened surface representing the conjunctiva.

To approximately 1 ounce of water in a clean rubber plaster bowl French's "regular" dental plaster is added with a spatula by being sprinkled into the water until only a slight film of clear water remains covering the settled plaster. The plaster is then mixed into a smooth homogeneous mass liquid enough to pour. Care must be given here to prevent the formation of bubbles by using a cutting stroke with the spatula and not a beating motion. The Müller lens with the film of negocoll is placed concave side up, and the liquid plaster is poured into

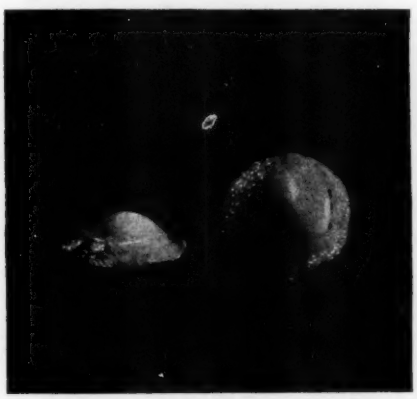

Fig. 15.—Plaster casts made from casting shells.

it. Before the plaster sets, a black thread, knotted at one end, is embedded in the plaster, with the knot at the side of the inner canthus as indicated by the earlier marking. If the plaster cast can be removed from the mold when hardened without destroying the surface of the mold, a second cast should be made at once for filing in the office. As soon as the cast is hard enough to write on, it should be marked with the patient's name or code number and some mark which will identify it as a cast of the left or the right eye. It is difficult to identify casts if they are left unmarked (fig. 13).

Ordering a Molded Contact Lens.—When ordering molded contact lenses a different procedure is followed from that used in ordering ground spherical lenses:

First, the cast takes the place of measurements of size.

Second, the patient's normal refraction as for spectacle lenses is given, plus the vertex distance of the refracting lenses from the cornea.

Third, the lenses are ordered oval; dimensions of 19 by 20 mm. are suggested, for these lenses must be placed on the eye in a definite position, in contrast to ground spherical lenses. It can be stated, however, that if they are placed approximately correctly they will naturally fall into their proper position.

Fourth, they must be marked "right" and "left" at the inner canthus to aid proper positioning. Because of the possibility of continued chemical reaction on the glass from the etching material, it is suggested that thin colored lines be fused on the outer side of the scleral band. A red line could be used for the right lens, which would easily be remembered for red and right (both begin with an "R"), and any other color could be used for the left inner canthus.

Fifth, in cases of keratoconus the mold is used for the scleral measurements. Refraction is determined with the best fitting spherical contact lens in place; this refraction and the radius of the corneal portion used are given on the prescription.

Sixth, two casts should be made, one to go to the Zeiss Works in Germany and one to be held for checking purposes.

Seventh, about two months instead of the usual five weeks is needed to complete an order for molded lenses.

CONCLUSIONS

Contact lenses have evolved over a period of more than one hundred years into thin, invisible practical aids to vision, comfortable to wear over periods of time. The Zeiss ground lenses are generally more satisfactory than the Müller type of lenses.

Indications for their use are irregularities and malformations of the cornea, ametropia, anisometropia, vocational and avocational activities that make spectacle lenses undesirable or dangerous, the cosmetic effect, the need for a protective covering for the cornea and protection from radiation.

The interest in these lenses centers today in the correction of the optic defects of the average patient with either a high or a low degree of ametropia.

The contraindications for their use are discomfort during the trial period, tight eyelids, hay fever or other allergic conditions, granular conjunctivitis, pinguecula and abnormalities and irregularities of the sclera and conjunctiva in addition to those mentioned.

Contact lenses should not be prescribed unless the patient can wear trial lenses for three hours unless there is pressure on the limbus which can be cleared with lenses with a continuous transition between the corneal and the scleral curvature.

Raised corneal portions are to be avoided whenever possible, and corneal sections with shorter radii should be substituted for them.

The most satisfactory method of fitting contact lenses is to fit the eye physically, disregarding refraction, and then determine the refraction of the patient with the contact lens in place on the eye. The required refractive power is ordered ground on the finished lens.

Refractive powers from —20 to +18 D. can be ground on the anterior corneal portion.

Much time and effort can be saved by making a cast of each eye during the first visit and fitting the contact lenses to these casts before the return of the patient.

Careful attention must be given to the corneal clearance, the fit of the scleral portion and the physiologic balance between the solution used with the contact lens and the patient's natural secretions.

The use of nitrazine test paper is a quick and accurate method of obtaining the p_H of the patient's lacrimal fluid.

In cases of low or moderate ametropia contact lenses must be worn in pairs. In cases of high ametropia, aphakia or keratoconus they can be worn successfully on one eye only.

Contact lenses have been worn for periods up to thirty-six hours, with no discomfort.

Patients with irregular scleras cannot wear spherical contact lenses comfortably.

Satisfactory casts can be made of the human eye with the aid of negocoll.

Blown contact lenses with ground corneal portions, entirely different from the Müller type of lens, can be made from casts after they have been modified in form. These blown lenses are reported as being comfortable to wear during the entire day.

Continued use and research with this type of lens may possibly result in the eventual use of this method to fit all contact lenses.

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News and Notes

EDITED BY DR. JOHN HERBERT WAITE

SOCIETY NEWS

Fifteenth International Ophthalmologic Congress.—For the convenience of those attending the Fifteenth International Ophthalmologic Congress, to be held at Cairo, Egypt, Dec. 8 to 15, 1937, four trips in the Orient have been organized under the auspices of the International Council of Ophthalmology, the parties leaving Marseilles on December 3.

The first trip comprises first class passage from Marseilles to Alexandria on the de luxe steamer Mariette Pacha, lunch and dinner on December 7 (the date of arrival at Alexandria), first class railway journey from Alexandria to Cairo and return, conveyance from the railway station to hotels and vice versa, stay at a first class hotel during the sitting of the congress (bed and breakfast only) and all meals on December 16 and 17, with arrival at Marseilles on December 21. Price: 1,620 belgas (about $275).

The second trip comprises the services indicated for the first trip up to December 16, with a visit to Upper Egypt (Luxor, the Valley of the Kings and Assuan, all expenses being included, with sleeping car accommodations) and return on the S. S. *Champollion,* with arrival at Marseilles on December 28. Price: 2,350 belgas (about $395).

The third trip will be the same as the second, with the addition of a visit to Palestine and Syria, with return to Marseilles on Jan. 4, 1938. Price: 3,400 belgas (about $575).

The fourth trip will be identical with the third except that the visit to Syria will be more complete (Homs, Hamah, Antioch, Latakia and other points of interest will be visited), with return to Marseilles via the northern Mediterranean (Rhodes, Smyrna, Istambul, Piraeus, Athens and Naples being visited). Price: 3,975 belgas (about $670).

The program giving full particulars of these trips will be sent free of charge on request from the Bureau des Croisières et Voyages Médicaux, 29 Boulevard Adolphe Max, Bruxelles, Belgium, the telegraphic address of which is Belgfranav, Brussels.

Philadelphia County Medical Society.—At the scientific meeting of the Eye Section of the Philadelphia County Medical Society, held on April 1, Dr. Mark J. Schoenberg addressed the group on "The Cooperative Study and Treatment of Glaucoma in Dispensary Patients for the Purpose of Reducing the Incidence of Blindness." Dr. B. Franklin Royer spoke as a representative of the National Society for the Prevention of Blindness.

GENERAL NEWS

Revue Internationale du Trachome.—The League Against Trachoma, the organ of which is the *Revue Internationale du Trachome,* which was originally founded by Morax, has now associated itself with the International Organization Against Trachoma. MacCallan, the

members of the League Against Trachoma and Bailliart have decided to place this journal at the disposal of the International Association Against Trachoma, and thus it will represent both societies. Articles in languages other than French will be accepted. The price of the journal, which is to be published every three months, will be 60 francs, payable through the Universal Postal Union. Dr. A. F. MacCallan, 33 Welbeck Street, London, W., England, is preparing a report on trachoma in various countries and will be grateful to receive references to the incidence of the disease in the following countries: Afghanistan, Albania, Armenia, Bolivia, Chile, Colombia, Costa Rica, Ecuador, Haiti, Honduras, Liberia, Nepal, Nicaragua, Panama, Paraguay, Peru, Salvador, Santo Domingo, Siam and Venezuela.

Summer Graduate Course in Ophthalmology and Otolaryngology, Denver.—The fifteenth annual summer graduate course in ophthalmology and otolaryngology will be held from July 26 to Aug. 7, 1937, at the Shirley-Savoy Hotel, Seventeenth Avenue and Lincoln Street, Denver, under the auspices of the Colorado Ophthalmological Society and the Colorado Otolaryngological Society. The following lectures on ophthalmology will be given:

"The Histology and Histo-Pathology of the Eye," Dr. Georgiana Dvorak-Theobald, Chicago.

"Recent Views of Glaucoma and Therapeutic Resources for Glaucoma," Dr. Andrew J. Browning, Portland, Ore.

"Congenital Syphilis," Dr. Arthur J. Markley, Denver.

"Normal and Pathological Eye Tissues," Dr. James M. Shields, Denver.

"Nonoperative Management of Cataract," Dr. Melville Black, Denver.

"Newer Operations for Strabismus," Dr. William M. Bane, Denver.

"Applied Physiologic Optics," Dr. Edward Jackson, Denver.

"Significance of Fields of Vision," Dr. Whitney C. Porter, Denver.

Further information regarding the complete program and the registration and fees may be obtained from Dr. H. L. Whitaker, 1234 Republic Building, Denver.

Course in Ophthalmology at George Washington University School of Medicine.—An intensive course in ophthalmology will be offered to graduates in medicine by the George Washington University School of Medicine from May 31 to June 5, 1937. There will be a symposium at the United States Army Medical Center (the Walter Reed General Hospital) on wounds of the globe, intra-ocular foreign bodies, sympathetic ophthalmia and ocular tumors. The National Bureau of Standards will devote one afternoon to the discussion of problems in the field of physics and optics allied to ophthalmology, such as color blindness, the study of optical instruments, spectroscopy, better lighting, polarimetry and the technic of glass making. The fee for the course will be $40. Further information may be secured from the secretary, Miss Louisa G. Wells, 927 Seventeenth Street, N. W., Washington, D. C.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

THE CILIARY INSERTION AND PROBABLE HISTOLOGIC SIGNIFICANCE OF THE ZONULAR FIBERS. G. BRUNO, Ann. di ottal. e clin. ocul. **64:** 400 (June) 1936.

Rabbit and human eyes were stained by various methods. The most posterior of the fibers were the most delicate; those most anterior were thicker (3 microns), being composed of a greater number of fine fibers. These fibers were continuous with fibers in the inner layer of the ciliary epithelium and may be compared to the cilia of other epithelial cells.

S. R. GIFFORD.

RELATIONSHIP BETWEEN THE SYMPATHETIC AND THE SHORT CILIARY NERVES. S. ERNYEI, Arch. f. Ophth. **136:** 40 (Sept.) 1936.

In the neighborhood of the short ciliary nerves Ernyei found a small sympathetic ganglion and bundles of nonmedullated nerve fibers which were a direct continuation of the sympathetic plexus of the internal carotid artery.

P. C. KRONFELD.

Bacteriology and Serology

ANTIGENIC PROPERTIES OF THE CORNEAL PROTEINS. S. RINALDI, Ann. di ottal. e clin. ocul. **64:** 649 (Oct.) 1936.

Rinaldi attempted to confirm the work of Kraupa, who found that the cornea possesses antigenic properties as shown by the complement fixation test. The author treated two dogs, three rabbits and two guinea-pigs with repeated injections of a corneal emulsion prepared with especial care as to freshness and sterility. For use as antigen in complement fixation tests a more concentrated emulsion was employed and also an alcoholic emulsion. In spite of injections of large quantities of the preparation made from the corneas of cows and of sheep and careful technic carrying out the complement fixation test, the results were all negative. It was also impossible to obtain anticomplementary tests with the material employed. Rinaldi concludes that the cornea possesses no antigenic properties or that these are so weak that they could not be revealed by the complement fixation test. He believes Kraupa's findings are to be explained by putrefactive changes and by the injection of bacteria with the material employed.

S. R. GIFFORD.

Biochemistry

CEVITAMIC ACID AND LENS METABOLISM. A. BAKKER, Arch. f. Ophth. **136:** 166 (Nov.) 1936.

In a previous communication (*Arch. f. Ophth.* **136:** 581, 1936) Bakker described a method of keeping an explanted lens alive for several days. The lens of a rabbit was put in a small glass container through which a slow steady stream of peritoneal transudate of the rabbit was run.

Lens and aqueous contain in vivo about ten times as much cevitamic acid as the blood, a fact which is difficult to explain. H. K. Müller believes that he has demonstrated that the lens can produce cevitamic acid. Goldmann and Buschke, on the other hand, assume that the reversibly oxidized form of cevitamic acid constantly diffuses from the blood into the aqueous. There the lens reduces it to the reduced form of cevitamic acid, which can penetrate the blood-aqueous barrier only with great difficulty. Thus may result the excess of cevitamic acid in the aqueous.

Bakker has carried out experiments on the surviving lens to prove or disprove the aforementioned assumption. He finds (1) that the capusle of the lens is freely permeable to both forms of cevitamic acid, (2) that the surviving lens cannot form cevitamic acid and (3) that even in the absence of cevitamic acid the rate of oxygen consumption and the transparency of the lens may remain normal.

P. C. KRONFELD.

Conjunctiva

THE DUST FACTOR IN THE PRODUCTION OF PTERYGIUM. T. J. DIMITRY, Am. J. Ophth. **20:** 40 (Jan.) 1937.

Dimitry does not believe dust to be the etiologic factor in pterygium. He shows that this condition is not so common in dusty sections of the country. Of all the geologic substances considered in a search for a particular agent, oil is the only one the geography of which parallels that of the pterygium. He feels that the question of etiology nears solution with the elimination of dust as the heretofore recognized cause.

W. S. REESE.

ANAPHYLACTIC ETIOLOGY OF ACUTE CONJUNCTIVITIS. G. FODOR and E. GLÜCK, Szemészet **70:** 260, 1935.

Bacteriologic investigations of the secretion of the conjunctiva revealed that in this regard there is but a slight difference between a normal and an acutely inflamed conjunctiva. In the case of acute conjunctivitis almost the same saprophytic micro-organisms are to be observed as in healthy eyes. Streptococci, staphylococci and pneumococci are the most frequent, while tubercle bacilli, Koch-Weeks bacilli and influenza bacilli are seldom noted.

So one must look for another cause of acute conjunctivitis. It is remarkable that the disease generally has its onset in the spring and

summer. Consequently we may consider other factors to be responsible —for instance, the effect of the ultraviolet rays of the sun, lymphatism and anaphylactic conditions of the organism. In the case of vernal conjunctivitis, several authors have tested the anaphylactic conditions of the organism against extract of pollen. It was pointed out that there exists hypersensitiveness against pollen in diseases of this type, for instance, hay fever, asthma and vernal conjunctivitis. The authors carried out experiments in the case of acute vernal conjunctivitis, using a polyvalent extract of pollen. As a result of such investigations anaphylaxis against pollen was found to play a marked rôle in the etiology of acute vernal conjunctivitis. Three quarters of the cases of so-called congestive catarrh were found to be cases of anaphylactic origin, and conjunctivitis of anaphylactic origin was found to appear usually binocularly. Vaccination was performed in fifty cases of acute conjunctivitis and in forty control cases.

N. BLATT.

Cornea and Sclera

RÔLE OF THE SYMPATHETIC NERVOUS SYSTEM IN THE GENESIS OF KERATITIS NEUROPARALYTICA. R. THUREL, Bull. Soc. d'opht. de Paris, October 1935, p. 618.

In collaboration with Cerise, 1931 Thurel reported on pathologic anesthesia of the cornea. Keratitis neuroparalytica, as its name indicates, is secondary to a nerve lesion which is carried not only by the sensory fibers of the trigeminal nerve but especially by the sympathetic fibers accompanying this nerve. Anesthesia of the cornea following destruction of the sensory fibers of the trigeminal nerve plays a rôle in the sense that the cornea, having lost its defense reactions, is exposed to noxious actions of external origin, both traumatic and infectious. Involvement of the sympathetic nervous system may be materially decreased by the protection afforded by blepharorraphy.

The case of a patient with recurrent attacks of keratitis neuroparalytica is cited to show that various noxious agents, such as tincture of iodine, may produce attacks in cases in which sympathetic section of the trigeminal nerve has been previously accomplished. Thurel also believes that herpes associated with inflammation of the trigeminal nerve is closely related to keratitis neuroparalytica and that both conditions are caused by an inflammatory reaction of the sympathetic nerves. Besides blepharorraphy, the use of automobile goggles is recommended for the cure of this condition.

L. L. MAYER.

MYCOSIS OF THE CORNEA AND MEIBOMIAN GLANDS DUE TO ACROSTALAGMUS. S. FAZAKAS, Magyar orvosi arch. **35:** 337, 1934.

A man 39 years of age exhibited thickening of the right lower eyelid. The thickening was most marked in the tarsus, especially at the line where the meibomian glands have their orifices. The intermarginal strip had enlarged to twice the normal size. The excretory

ducts of the meibomian glands were markedly dilated and tortuous. From these orifices a seropurulent fluid was discharged in which, even with the naked eye, small granules were visible. The latter were identified on microscopic examination as pure cultures of fungi. The lower portion of the cornea presented yellowish gray globular infiltrations the size of a pinhead, equal in size and with a stippled surface. These infiltrations ran a protracted course and resulted in the confluence of several infiltrations. From the secretion of the meibomian glands and the detritus of the corneal scrapings the fungus Acrostalagmus cinnabarinus could be cultured. Inoculations into animals gave uniformly positive results. Six or seven days after the inoculation had taken place the cornea of the rabbit exhibited a typical picture of mycotic keratitis. Retroculture from the cornea of the rabbit also produced a positive result. Acrostalagmus cinnabarinus is therefore pathogenic not only for the skin but also for the cornea. N. BLATT.

Experimental Pathology

ACTION OF AVITAMINOSIS C ON THE POTENTIAL OF AQUEOUS AND LENS. N. BEZSSONOFF, J. NORDMANN and P. REISS, Compt. rend. Soc. de biol. **123:** 1196, 1936.

The oxidation-reduction potential of the aqueous and of the lens of guinea-pigs was determined before and after dietary deprivation of vitamin C. In all cases avitaminosis C resulted in an increase in potential, producing an average rise of 60 millivolts in aqueous and 40 millivolts in lens.

J. E. LEBENSOHN.

CULTIVATION EXPERIMENTS WITH FUNGI OF THE CONJUNCTIVA, CORNEA, PALPEBRAL MARGIN AND LACRIMAL DUCT. S. FAZAKAS, Magyar orvosi arch. **35:** 325, 1934.

The conjunctival sac and the lacrimal passages constitute a good medium for fungi, owing to the constant humidity, the heat and the protection from direct rays of the sun. The globulin content of the tears, too, increases the value of these parts as culture mediums. These theoretical reflections induced Fazakas to examine fungi possibly present on the anterior segment of the eye by cultures on a suitable medium. He considered the results as positive when the respective type of fungi had developed on several soils at the same time. As mediums, Sabouraud's maltose agar and Benedek's culture medium (dextrose and peptone) were used.

The investigations revealed a remarkable frequency of fungi in the various diseases of the eye. In diseases of the conjunctiva all sorts of genera and species of fungi were found in 157 cases; in diseases of the cornea a fungus could be traced 39 times, in diseases of the palpebral margin, 118 times, in diseases of the lacrimal passages, 41 times and in normal eyes, 39 times. Among 1,017 diseased eyes fungi could be identified in 365 (35.8 per cent); among 160 healthy eyes, however, they could be identified in only 39 (24.3 per cent). The rate of incidence was greatest for Penicillium (in 36.30 per cent

of the cases), while for Torula it was 12.68 per cent, for Alternaria 5.22 per cent, for Scopulariopsis brevicaulis 4.97 per cent, for Aspergillus 4.47 per cent, for Haplographium 6.71 per cent, for Hormodendrum 3.73 per cent and for Cladosporium 3.48 per cent. As a result of such investigations it is concluded that fungi present in the anterior segments of the eye are supposed to play a pathogenic part in diverse diseases of the eye.

N. BLATT.

General

MISTAKES IN THE DIET IN OPHTHALMOLOGY. M. KÖTELES, Szemészet **70:** 202 (Sept. 30) 1935.

It is generally known that nutrition exerts a great influence on the human organism. In the last century, in the conception as to nutrition greatest stress was laid on caloric values, while at present a new factor of nutrition, the vitamins, has become most important. In the domain of diseases of the eye avitaminosis plays a great rôle. In accordance with observations up to the present, vitamin A has been of most importance for the eyes. If vitamin A is missing in the food, hemeralopia may occur, and anatomic changes in the eye may follow. According to more recent investigations at the ophthalmic clinic of Basel, Switzerland, the aqueous of the eye contains the greatest percentage of vitamin C. Experiments made on scorbutic guinea-pigs revealed the fact that with intense avitaminosis the lens becomes clouded and cataract often occurs. Consequently there should be a connection between the pathologic aspects of the formation of cataract and avitaminosis C of the organism. In treating incipient cataract a diet abundant in vitamin C is provided. Concerning the question of bacilli found in xerosis it is reported that in avitaminosis A the vitality of the eyes declines, and thus the virulence of the micro-organisms is increased. The treatment of avitaminosis starts with prophylactic measures. In the case of growing children special care should be taken to secure food rich in vitamins. If xerophthalmia (atrophy of the conjunctiva and cornea) or keratomalacia (softening of the cornea) is present, the diet prescribed should abound in vitamin A, along with adequate doses of sodium chloride. At the same time local treatment of the eye should be carried out. There are other disturbances of nutrition which are not caused by avitaminosis. Pellagra is due to tainted corn. Pellagra may give rise to hemeralopia, optic neuritis, choroidal hemorrhage, defects in the visual field or in the color sense, asthenopia, diplopia and cataract. Inequality of the pupils, mydriasis and abnormal contraction of the pupils may sometimes occur. An appropriate diet is therefore of special therapeutic importance for patients with diseases of the eye. If the disease of the eye occurs in connection with any disturbance of metabolism, the diet should be appropriate for the primary disease (diabetes, gout, arteriosclerosis, nephritis, leukemia, etc.). A light diet, especially one consisting of liquid and soft food, is generally prescribed after operations on the eye.

N. BLATT.

General Diseases

SOME OCULAR SYNDROMES OF HEPATIC ORIGIN. V. ROSSI, Arch. di ottal. **43:** 175, 1936.

Rossi compares the liver with the eye in respect to certain functions and discusses certain conditions in which a derangement of both tissues may be supposed to exist. Probably the most important of these conditions are the disorders of lipoid metabolism. No cases are reported.

S. R. GIFFORD.

EVALUATION OF PULMONARY FINDINGS IN OCULAR DISEASES OF SUSPECTED TUBERCULOUS ORIGIN. W. ROHRSCHNEIDER and H. REINERS, Klin. Monatsbl. f. Augenh. **96:** 778 (June) 1936.

Rohrschneider and Reiners undertook to solve the following questions: 1. How frequently are tuberculous pulmonary changes associated with inflammatory diseases of the uvea, and to what extent may these changes be used in establishing the etiology of the ocular disease? 2. Is it possible to determine whether in association with ocular diseases there are pulmonary conditions which are clinically strongly suggestive of tuberculosis and others which are only slightly suggestive of tuberculosis?

Exact clinical and roentgenologic examinations of the lungs of seventy-two adults suffering chiefly from inflammation of the uvea showed only minor and closed remnants of tuberculous processes in sixty-four. In six others a pulmonary process was found which had formerly been active but which was completely inactive at the time of the observation.

The remaining two patients presented active pulmonary tuberculosis, requiring treatment. No difference in the pulmonary condition was noted in the cases of ocular disease associated with clinical signs of tuberculosis and in the cases of ocular disease associated with less suspicious signs of tuberculosis.

The authors evaluated frequent but clinically irrelevant pulmonary changes associated with these ocular diseases as etiologically less important than other authors recently did. Similar pulmonary changes were found rather regularly in healthy adults. Therefore, a reliable conclusion regarding the tuberculous etiology of the ocular disease is impossible. Hence Marchesani's opinion that the tuberculous etiology of chronic uveitis is not as well established as has been assumed by many writers is correct.

K. L. STOLL.

CHANGES OF THE EYE IN SYPHILIS OF LONG STANDING. A. I. VOLOKONENKO, Sovet. vestnik oftal. **9:** 342, 1936.

One hundred and forty-two patients who had primary syphilitic infection from ten to twenty years previously, were examined by an ophthalmologist, an internist, a neurologist and a syphilologist in order to ascertain the influence of the treatment on the syphilitic process.

Fifty-two per cent of the patients were aged from 20 to 39 years, and the others were aged up to 60. Twenty-eight and three tenths

per cent had had no treatment for syphilis when they first came for an examination; 63 patients had had early and prolonged treatment, and 71 had had insufficient treatment. In 52 patients, or 39 per cent, no changes in the eye were found; of these, 59 per cent received constant treatment. In 7.6 per cent of the patients with syphilis who had been treated there were changes in the eyes; of the patients who had received poor treatment for syphilis, changes in the eye were observed in 19 per cent.

The eyes were affected in the following manner: Optic atrophy was present in 8 patients; perivasculitis and retinal hemorrhage in 1, and small choroidal lesions at the periphery of the fundus in 13. Pupillary changes were noticed in 29.8 per cent of the patients.

The conclusions of this study are as follows:

1. Changes in the visual organ can be avoided by proper and early treatment of syphilis.

2. Antisyphilitic treatment started in the second stage of the disease does not prevent the appearance of lesions in the eyes.

3. Changes in the visual organ were observed in 17 per cent of the patients who received late or insufficient treatment for syphilis.

4. The choroidal changes had a characteristic ophthalmoscopic picture.

5. Pupillary changes were observed in 29.8 per cent of the untreated patients.

O. SITCHEVSKA.

Glaucoma

PATHOGENESIS OF HYDROPHTHALMOS. A. SANTONOSTOSO, Ann. di ottal. e clin. ocul. **64:** 405 (June); 437 (July) 1936.

The literature on the pathogenesis of hydrophthalmos, especially that dealing with the histopathologic aspects, is reviewed. Thirteen cases are reported, including one in which the condition was studied histologically at an early stage. The patient was a child of 5 years whose mother had been treated for syphilis during lactation. The right eye had begun to increase in size at the age of 4 months. The Wassermann reaction of the blood was positive for the mother and the child. The corneal diameter measured 17 mm., and the tension was 52 mm. Vision was nil. The left eye was normal. Section of the right eye showed absence of Schlemm's canal, with the usual changes due to distention of the globe. There were slight inflammatory changes, with infiltration of the iris and ciliary body and marked endarteritis of all the vessels. In nine of the cases the condition was bilateral, and in nine it occurred in males. In one case in which the hydrophthalmos was bilateral a vascular nevus of the face was present on one side, while in one case in which the hydrophthalmos was unilateral it was evidently secondary to interstitial keratitis. Among four cases in which a Wassermann test was made, in only one was there a positive result, but in five cases it was considered that syphilis was probably present in the parents or children.

Santonostoso believes syphilis to be an important cause of hydrophthalmos. In his case sections indicated that a canal of Schlemm may

have been present but was obliterated by inflammatory changes, and he considers that the findings in other reported cases of hydrophthalmos may be interpreted in the same way. Photographs, photomicrographs and a bibliography accompany the article.

S. R. GIFFORD.

CHOICE OF OPERATION IN GLAUCOMA. V. P. STRAKHOFF, Sovet. vestnik oftal. **9:** 623, 1937.

Strakhoff first discusses in detail the early diagnosis and the conservative treatment of glaucoma and then takes up the various operative methods for various forms of glaucoma. From his vast experience he has come to believe that miotics can be applied safely in all cases of glaucoma in which the function of the eye and of the optic nerve has not been impaired.

He uses iridectomy in acute inflammatory glaucoma only, after the tension has been brought down with miotics. Elliot's trephine operation is used in all cases of chronic simple and chronic inflammatory glaucoma in which fairly good visual fields are preserved. Cyclodialysis is the operation most frequently used by the author, as he found it to be effective even in patients with very contracted fields and with only one eye.

In cases in which the results of cyclodialysis were not successful he employed as an additional measure simple sclerotomy,

Case histories illustrate the author's point in favor of one or another operation.

O. SITCHEVSKA.

Injuries

SPONTANEOUS EVACUATION OF INTRA-OCULAR LEAD SHOT. G. SZABÓ, Szemészet **70:** 135 (Sept. 30) 1935.

Perforating wounds from lead shot are nearly always fatal for the eye; in most cases not only is vision lost, but shrinking of the eyeball results. With such injuries the fate of the eye depends on three factors: (1) the location and size of the wound, (2) the question of where the shot remained in the eye and (3) the matter of septic infection, in particular. It is known, however, that in the case of bullet wounds purulent infections seldom occur. Chemical effects are also of importance for the outcome. Particles of iron and copper are chiefly dangerous owing to the possibility of siderosis or chalcosis. The eye does not tolerate copper and iron and tends to eliminate such particles spontaneously. On the other hand, there is considerable tolerance of the eye for lead, intra-ocular particles of lead or lead shot being expelled in rare cases only. One case of this type is related by the author. A man of 34 was hurt while hunting. Seventeen years later a vesicle-like eruption appeared below and close to the limbus of the wounded eye, which had extremely poor vision. Roentgen examination gave evidence of the foreign body, which was removed through a superficial incision. In addition to this there were a large choroidal retinal scar at the fundus, denoting double perforation of the bulbus, and a membranous cataract, which ultimately was removed by operation.

N. BLATT.

Lens

CATARACT EXTRACTION WITH SUCCEEDING EXTRACTION OF THE CAPSULE. B. VON HORVÁTH, Klin. Monatsbl. f. Augenh. **96:** 746 (June) 1936.

Horváth gives a detailed and well illustrated description of his new method of extracting cataracts. After performing an iridectomy he makes a large incision with Rosas' discission needle in the capsule of the lens near the equator and parallel to it. After delivery of the cataract the capsule is grasped near the lower pupillary margin in such a way that one prong enters the bag while the other enters the anterior chamber between the bag and the cornea. The corneal flap is lifted off by a forceps during this procedure. In some cases a second forceps is used after a portion of the bag has appeared outside the wound; in other cases the extraction is completed by means of a blunt iris hook.

The disadvantage of this operation is that particles of the cortex may remain in the eye, and iridectomy is required. K. L. STOLL.

IS MEDICAMENTOUS TREATMENT OF SENILE CATARACT POSSIBLE? H. K. MÜLLER, Klin. Monatsbl. f. Augenh. **97:** 304 (Sept.) 1936.

Müller refers to the views of Vogt, Greeff and Elschnig, who denied that senile cataract may be cured or prevented by medicamentous treatment, and to Römer and Siegrist, who attempted such a cure but failed. Nevertheless, Müller, for reasons given in the paper, arrived at the conclusion that the thought of giving medicamentous treatment for senile cataract cannot be dismissed. The metabolism of the albumin and carbohydrate in the lens is referred to; the results of research on glycolysis, done by the author and other writers, are analyzed, and the research on the high content of glutathione, vitamin C and vitamin B_2 in the lens is discussed. Personal research showed Müller that certain poisons influence the glycolysis and the vitamin content in the lens. In the course of his argument he stresses the anatomic and physiologic conditions which cause the lens to be practically an independent and isolated portion of the body. K. L. STOLL.

Methods of Examination

THE COLLOIDOMETER, AN APPARATUS TO MEASURE THE LIGHT PASSING THROUGH THE ANTERIOR CHAMBER: REPORT OF CASES. HENNING RÖNNE, Klin. Monatsbl. f. Augenh. **97:** 161 (Aug.) 1936.

The rays of light of the slit lamp passing through the refracting media of the eyeball produce an illuminated area (*Lichtweg*) in the cornea and lens in accordance with Tyndall's law. These two *Lichtwege* are connected by a fainter illuminated area crossing the anterior chamber whenever its albumin content is increased as a result of ocular inflammatory processes. The appearance of this *Lichtweg* in iritis is probably the most important clinical symptom brought to notice by the slit lamp, in Rönne's opinion. The strength of Tyndall's phenomenon depends on the size of the colloid particles in the anterior chamber. Rönne

devised a disk which is attached to the slit lamp in such a manner that the light passes through a series of slides of gray glass of varying density. This attachment, called the colloidometer, allows exact readings of the albumin content and its variations in the course of the treatment. It was used with good results in cases of iritis and cases of sympathetic ophthalmia and after extraction of complicated and other forms of cataracts. Differences were observed in the albumin content in "rheumatic" and tuberculous iritis. The albumin content in the aqueous, formed after paracentesis of the anterior chamber, was increased only slightly or not at all. Rönne has used his colloidometer for the past three years. Two cases are reported to illustrate the variation of the albumin content from the beginning of the treatment to the completion of recovery.

K. L. STOLL.

Neurology

ARACHNOIDITIS AT THE OPTIC CHIASM. Q. DIMARZIO, C. CAVINA and G. DENIGRIS, Riv. oto-neuro-oftal. **13**: 291 (July-Aug.) 1936.

Seven cases of arachnoiditis at the optic chiasm are described. Some of the cardinal signs, such as sudden onset of loss of vision, hemianopia and edema of the disk followed by optic atrophy, are discussed. The authors make a point of the fact that in edema of the disk caused by serous meningitis optic atrophy takes place slowly while in cases of arachnoiditis of the type described the change from edema to atrophy is sudden.

They advise operation in every case in order to save the patient's sight.

G. BONACCOLTO.

BLINDNESS DUE TO METASTATIC SYMMETRICAL ABSCESSES OF THE OCCIPITAL LOBES AND RIGHT HEMIPLEGIA CAUSED BY ABSCESSES OF THE LEFT PARIETAL LOBE. A. TOMMASINI-MATTIUCCI, Riv. oto-neuro-oftal. **13**: 329 (July-Aug.) 1936.

In this case the value of the classic physical signs are discussed: blindness, absence of ocular findings and presence of light reflexes. The diagnosis of abscesses of the occipital lobes was confirmed by autopsy.

G. BONACCOLTO.

ACUITY OF VISION FOLLOWING DECOMPRESSION TREPHINING FOR CHOKED DISK DUE TO A CEREBRAL TUMOR. D. LAZARESCU, Rev. stiint. med. **22**: 833, 1933.

Twelve patients who had suffered from cerebral tumor associated with choked disk underwent decompressive craniectomy.

Postoperatively vision improved in one case only and to a very small extent. In the other cases vision remained at zero, as before operation, or if vision was present preoperatively it became zero after operation.

The postoperative mortality was 83.33 per cent. The patients died three days after operation at the earliest and up to one year at the latest.

In not a single case did the tension of the cerebrospinal fluid become normal following decompressive craniectomy.

N. BLATT.

Orbit, Eyeball and Accessory Sinuses

INCLUSION OF GLASS OVOIDS IN THE SCLERAL CAVITY. E. MORETTI, Ann. di ottal. e clin. ocul. **64:** 661 (Oct.) 1936.

Moretti practiced a modification of the Mules operation as employed by Poulard. The use of a glass ovoid with its long axis parallel to the palpebral fissure was preferred to the use of a glass ball, since often a reform eye could not be worn over the latter. The cornea was apparently excised, and the sclera and conjunctiva were sutured over the implant. Twenty-five operations were performed, including six on patients with panophthalmitis. The implant was retained, with excellent results, in nineteen cases. In three of the cases in which failure resulted the ball was ejected on the day after the operation, while in the other three its loss occurred from seven to ten days later. In cases in which the ball was lost the outcome is the same as that following ordinary evisceration. The motility and cosmetic effect apparently left nothing to be desired in the cases in which the procedure was successful.

Moretti advises that the operation be performed as soon as possible in cases of panophthalmitis, in which the chances are best that the implant will be retained. The procedure was successful in five of the cases of panophthalmitis.

S. R. GIFFORD.

The Pupil

CONTRACTION OF THE PUPILS ACCOMPANYING FOCUSING FOR NEAR VISION. H. BURIAN and G. SCHUBERT, Arch. f. Ophth. **136:** 377 (Dec.) 1936.

For the reaction of the pupils which accompanies convergence or accommodation Behr suggested the noncommittal term "reaction accompanying the focusing for near vision" (hereinafter designated as the FN reaction) because "despite an immense amount of work it is still undecided whether the FN reaction is coupled with the process of accommodation or with that of convergence." During the last eight years, however, more and more evidence in favor of the view that the FN reaction is a reaction to convergence has been collected. Burian and Schubert report haploscopic experiments in which the pupils did not contract when the accommodation changed from 0 to 8 diopters (haploscopy with parallel visual axes). A convergence of 3.5 degrees, however, promptly elicited a contraction of the pupils. The FN reaction, therefore, is a reaction to, or accompanying, convergence.

In another series of haploscopic experiments the authors studied the dioptric effect of the FN reaction. Using Gullstrand's subjective stigmatoscopy (Tigerstedt, R.: Handbuch der physiologischen Methodik, Leipzig, S. Hirgel, 1911-1912, vol. 3, pt. 1, p. 118) they found that during haploscopic near vision with parallel visual axes the size of the pupil exceeded its optical zone. During the FN reaction, however, the size of the pupils was smaller than that of the optical zone. Furthermore, the resolving power of the eye was greater during the FN contraction of the pupils. Thus the FN reaction provides a greater

resolving power and a greater acuity in perception of depth. Besides, this reaction increases the range of stereoscopic near vision or, as it were, the depth of the horopter. No definite statements can be made with regard to the central nervous mechanism which links up convergence and the FN reaction.

P. C. KRONFELD.

Physiology

COURSE OF DARK ADAPTATION WITH LOW OXYGEN TENSION. E. BUNGE, Arch. f. Augenh. **110:** 189, 1936.

The course of dark adaptation of seven normal persons under the influence of low oxygen tension was investigated. The subject was examined while breathing through an apparatus designed to give artificial respiration at normal barometric pressure, and in the majority of experiments the minute volume was regulated to 10 liters containing 10 per cent oxygen. Bunge could find no influence of the lack of oxygen on the regeneration of the visual purple.

F. H. ADLER.

BINOCULAR PERCEPTION OF THE DEPTH OF VERTICAL LINES OF WHICH EACH EYE SEES ONLY ONE (UPPER OR LOWER) HALF. H. BURIAN, Arch. f. Ophth. **136:** 172 (Nov.) 1936.

F. C. Donders and his associates were the first to report that perception of the depth of falling balls could be recognized properly if the upper half of the field in one eye and the lower half in the other eye were shut off by "hemianopic" diaphragms. Burian, under the direction of Tschermak, Prague, has studied this form of perception of depth very thoroughly on the horopter apparatus. The results do not lend themselves to abstracting.

P. C. KRONFELD.

RELATION BETWEEN VASCULAR PRESSURE IN THE RETINA AND CEREBRAL PRESSURE MEASURED WITH THE AUTHOR'S APPARATUS. F. KUKÁN, Klin. Monatsbl. f. Augenh. **97:** 331 (Sept.) 1936.

The authors who have done research on the relation of the vascular retinal pressure to the cerebral pressure may be divided into two groups. The larger group maintains that the arterial pressure is decisive, while the smaller group, including Baurmann and Sobánski, are convinced that a systematic connection can be obtained only by finding the venous pressure. Kukán agrees with the second group. Tables contain records for thirty-four patients. They show the medical diagnosis; the condition of the fundus; data for the cerebral pressure, found by measuring the pressure in the central retinal vein with Baurmann's apparatus and Sobánski's apparatus; data for the lumbar pressure, the pressure in the brachial artery and the diastolic pressure in the central retinal artery. Kukán discusses the content of these tables in detail and arrives at the following conclusions: A decided parallelism has been observed between the pressure in the retinal vein and the lumbar pressure. When Baurmann's method of calculation was employed, the cerebral pressure could always be deducted from the

pressure in the retinal vein, and the lumbar puncture corroborated the result. Increased cerebral pressure was present in every case in which increased pressure of the cerebrospinal fluid was evidenced by increased venous pressure. Kukán expects that his ophthalmodynamometer and his method, which are based on the method of Baurmann and that of Sobánski, will yield still more accurate values.

K. L. Stoll.

Refraction and Accommodation

A New Visual-Acuity and Astigmatism Test Chart. C. E. Ferree and G. Rand, Am. J. Ophth. **20:** 21 (Jan.) 1937.

Ferree and Rand discuss the requirements of a good test chart and the disadvantages of test objects of the capital letter type. They present a chart in which double broken circles are used. This can be used for testing acuity and astigmatism.

W. S. Reese.

Invisible Contact Glasses. J. Dallos, Szemészet **70:** 219 (Sept. 30) 1935.

This paper deals in general with the tactile and optic effects of contact glasses. The idea of contact glasses originated with the physicist Herschel and dates from 1827. The idea was carried on by Fick, an ophthalmologist of Zurich, Switzerland, who tried to correct particularly irregular forms of astigmatism by means of contact glasses. August Müller, who tested the effect of such glasses on his own eyes, asserted that the new artificial "corneal-lens" was unbearable. Blown contact bowls made by Müller of Wiesbaden were far better tolerated by the eyes but were said to be limited to improving the acuteness of vision. On the other hand, contact glasses ground by Zeiss of Jena are said to be ideal in optical corrective effect, though unbearable. It is desirable that a combination be invented which will combine the tactile advantage of the blown glasses with the ideal optical effects of ground contact glasses.

According to Dallos, this result may be attained if an impression is first obtained from the living eye. Contact glasses constructed after such impression molds work ideally in both a tactile and an optical way. The author is said to have succeeded in making a cast by using Poller's negocoll. This plastic material is put into a contact glass of fairly good fit and used for the impression. From this negative cast the positive cast is made of another material, Poller's homonit. An accurately fitting glass bowl is made from this. The dioptric effect of such contact glasses is corrected by grinding the front central part. Contact glasses are indicated in cases of irregular astigmatism, high ametropia or anisometropia, to serve as a substitute for spectacles the use of which is impossible or inadvisable for professional reasons, as protective glasses in the case of entropion with turning in of eyelashes and widening of the palpebral fissure, and for intensely weak-sighted patients, as a telescope in combination with convex spectacles.

N. Blatt.

Retina and Optic Nerve

THE SURGERY OF RETINAL DETACHMENT. H. S. GRADLE and S. J. MEYER, Am. J. Ophth. **19:** 873 (Oct.) 1936.

Gradle and Meyer review their experiences with the Gonin, Larsson, Guist and Weve methods of operating for retinal detachment and the method of operating with the single short needle. They give their impressions and state the following conclusions:

"The modern methods of inducing adhesive choroiditis by diathermy puncture of the vitreous with a resultant watertight annular barrier around the hole or disinsertion offer the best prognosis in present-day surgery of detachment of the retina.

"The indications for operation are still somewhat sketchy and definite contra-indications have not yet been formulated. As a result we are still justified in operating in every case of retinal detachment that offers any possible hope.

"If practically all eyes with retinal detachment are operated upon by modern methods, reattachment will take place in somewhat less than fifty percent. But if only selected eyes are operated upon, the percentage of reattachments will be nearer seventy." W. S. REESE.

A CASE OF PERIVASCULITIS RETINAE ASSOCIATED WITH SYMPTOMS OF CEREBRAL DISEASE. A. J. BALLANTYNE and I. C. MICHAELSON, Brit. J. Ophth. **21:** 22 (Jan.) 1937.

A man aged 30 years gave the following history: For eight years he had been having fits once in two months. Before each attack his face twitched and everything became black. The attack was followed by sleepiness, but this was common at all times, and he had even fallen asleep at his work. There was a tendency to catatonia.

The patient complained of dimness of vision of the left eye. There were hemorrhages in the vitreous. The veins in some parts showed parallel sheathing. There were many retinal hemorrhages. Two weeks later there was a large hemorrhage in the vitreous and in the right eye there were exudates and hemorrhages. The retina in each eye showed pronounced varicose vessels in the papillary zone, and the straight vessels were injected.

Later, secondary glaucoma developed in the left eye, and this eye was excised. The results of physical examination and laboratory tests were practically negative except for a positive von Pirquet reaction to human and bovine tuberculin. The histologic observations are given in detail.

The authors' summary and conclusions are as follows:

"A case of perivasculitis retinae has been clinically and pathologically described.

"A group of 68 similar cases reported in the literature has been discussed with regard to the clinical facts, the pathological appearances, the aetiology, the path of entrance of an infection, the implication of the cerebral vascular system, and the association with recurring vitreous haemorrhages.

"The pathology of perivasculitis retinae in young adults is an inflammatory cellular exudate within the vessel walls, and usually confined to the veins.

"A similar condition probably affects some of the intracranial vessels.

"Perivasculitis retinae probably frequently manifests itself in the clinical condition described as recurring haemorrhage in the vitreous of adolescents.

"The cause is not definitely determined although a good percentage of cases are associated with active tuberculosis.

"The case which we report appears to be due to an unknown infection.

"It is possible that perivasculitis retinae is not the expression of a single disease, but rather a notable clinical phenomenon common to several diseases, of different aetiology and distinguished from each other clinically by such features as differences of age incidence, localization of the changes in the veins alone or in both arteries and veins, the more or less peripheral situation of the early lesions, the rapidity of the pathological process, the occurrence of vitreous haemorrhage, and the presence of disturbances of the central nervous system."

The article contains a number of illustrations, including a colored plate.

W. ZENTMAYER.

REPEATED OBLITERATION OF THE CENTRAL ARTERY OF THE RETINA. S. SCHIFF-WERTHEIMER and H. BALLARD, Bull. Soc. d'opht. de Paris, October 1935, p. 615.

The case of a patient with a curious clinical history is reported. Sudden blindness occurred and persisted for several minutes. Recovery was complete, but the retinal arterial tension was lowered. The attack was usually preceded by pain about the left orbit and hemicrania of the same side. Edema of the papilla and retina, involving the macular region of the left eye, together with arterial narrowing, was noted on ophthalmoscopic examination. The left pupil increased in size during the attack, and the visual field was concentrically contracted to 20 degrees. Intramuscular injections of acetylcholine brought about a return of the fundus, the fields and vision to normal. During one month the attacks occurred four times. General examination, including a Wassermann test of the blood, failed to reveal evidence of disease, except for rheumatism. A diagnosis of repeated spasm of the central retinal artery was made.

L. L. MAYER.

DIATHERMOTHERAPY OF RETINAL DETACHMENT. L. MAGGIORE, Ann. di ottal. e clin. ocul. **64:** 361 (June) 1936.

Maggiore reviews the literature and draws certain conclusions from his own experience with the method. In two cases he observed spontaneous reattachment, and in one of these he followed the result for years. He calls attention to cases in which the retinal discontinuity, as he prefers to call the tear, was present in an area of retina removed

from the detachment. He believes that scleral coagulation and coagulation with pins are each suitable in certain cases, while in others a combination of both methods may be useful. In cases without any visible discontinuity he coagulates so as to wall off the detached from the normal retina. In some cases one such operation was successful, while in others detachment subsequently developed in other areas, requiring two or three operations, but with final success. One such case in an aphakic eye is reported. While in most cases the usual prolonged rest in bed was prescribed after operation, in two cases the patients were allowed to go home immediately after operation and obtained good results. In only one case in his series was hemorrhage into the vitreous a serious complication. Return of vision depended on the duration of the detachment and on whether the macular region was detached. In one case a medical colleague reported restoration of normal vision at the conclusion of the operation. From the literature and his own material Maggiore concludes that good results are to be expected in from 65 to 70 per cent of the cases. Among factors unfavorable to success are long duration of the detachment, old age and an incipient cataract which makes observation of the fundus difficult. One good result was obtained in a detachment of two years' duration, although the result was not followed long. Marked hypotony is an unfavorable condition, as is the presence of dense vitreous opacities. In aphakic eyes he has obtained one success and one failure. S. R. GIFFORD.

RESULTS OF SURGICAL TREATMENT OF RETINAL DETACHMENT. F. RAMACH, Arch. f. Ophth. **136:** 78 (Sept.) 1936.

Ramach reports the final results obtained in the cases of retinal detachment in which operations were performed at Lindner's Clinic during the years 1932, 1933 and 1934 (follow-up examinations were made from one and one-half to two years after the last operation). Four surgical methods were used: multiple trephining followed by cauterization of the exposed choroid with potassium hydroxide after Guist, the undermining operation with injection of a solution of potassium hydroxide into the suprachoroidal space after Lindner, diathermic coagulation after Weve and Šafář, and the excision of strips of sclera after Lindner. The cases were classified as uncomplicated and complicated, following the suggestion of J. Visser-Heerema and Weve (*Klin. Monatsbl. f. Augenh.* **94:** 721, 1935), the criteria of complicacy being: aphakia or dislocation of the lens, a tear in the macula, pronounced nystagmus, severe congenital anomalies and glaucoma. Cases in which operation was performed several times and after different methods were listed according to the type of operation performed first. The results obtained with undermining were better than those obtained with the other methods. The grand total shows that in 50 per cent of all the cases complete reattachment lasting for at least one and one-half years from the time of the last operation was accomplished. This percentage of cures is not quite as high as the percentage in previous years, probably because of the large proportion of persons with complicated detachments, uncomplicated detachments being more frequently operated on in smaller

hospitals. The chances for reattachment after operation would be better if in every case of detachment, as soon as the diagnosis is made, pinhole spectacles (Lindner) were ordered.

Method	Patients Operated On	Patients Cured	Patients with Uncomplicated Detachments: Operated On	Patients with Uncomplicated Detachments: Cured
Multiple trephining, KOH cauterization (Guist)...	15	7 = 46.7%	13	7 = 53.8%
Undermining (Lindner).....	71	39 = 55.0%	44	29 = 66.0%
Diathermy (Weve, Šafář).....	62	28 = 45.1%	43	23 = 53.5%
	148	74 = 50.0%	100	59 = 59.0%

P. C. KRONFELD.

DETACHMENT OF THE RETINA DURING PREGNANCY. MARTIN GOERLITZ, Klin. Monatsbl. f. Augenh. **97:** 22 (July) 1936.

A woman suffered spontaneous detachment of the retina of the right eye during the fourth month of her fifth pregnancy. Her general physical condition had been perfect, and vision of the right eye was normal after correction of myopia of 7 diopters. The left eye had vision of only 6/18, with myopia of 8 diopters and astigmatism of 3 diopters. The detachment was arc-shaped and located at about 12 o'clock; there was a gaping tear. Opacities of the vitreous were absent. Interruption of the pregnancy was advised by a consultant, but Goerlitz performed a coagulation operation after the method of Weve. The deeply reclining position had to be discontinued on the day after the operation, when the patient complained of pain in the region of the kidneys. Urinalysis showed albumin (0.1 per cent) and a few cylinders. The urine was normal after eighteen days, and the subjective symptoms disappeared. The detached portion of the retina was completely reattached when the patient was dismissed on the twenty-eighth day after the operation. Vision in the eye on which the operation was performed was 6/9 about fifteen months later. The confinement terminated in a normal manner, without any ill effects on the health of the mother or the child. Goerlitz concludes that detachment of the retina in a highly myopic eye is no indication for interruption of pregnancy in the absence of retinal or other symptoms of toxicosis.

A number of additional cases are cited by Goerlitz illustrating his results and his technic in operating on highly myopic eyes.

K. L. STOLL.

TOTAL COLOR BLINDNESS, HEMERALOPIA AND TAPETORETINAL DEGENERATION IN TWO SISTERS: GENEALOGY AND REPORT OF CASES. E. BUNGE, Klin. Monatsbl. f. Augenh. **97:** 243 (Aug.) 1936.

Bunge found the following conditions in two sisters in 1935. The elder sister, born in 1911, presented photophobia, jerking nystagmus, bilateral keratoconus, with a corneal radius of 5.7 mm. in the right eye and 5.5 mm. in the left, irregular astigmatism in both eyes and a few radii in the cortex of the left eye. A scarcity of choroidal pigment was noted in both fundi, the choroidal and vorticose veins being bared. The yellow of the macula lutea was barely noticeable with red-

free light. Vision of each eye was greatly reduced, and the field of vision, which was obtainable in a dimly lighted room only, was normal when taken at a distance of 33 cm.

Reduced sensitiveness to light, hemeralopia and total color blindness were evidenced by several tests. The syndromes noted in this case have never been described in the literature.

The younger sister, born in 1917, presented similar symptoms, but some of them were less severe. In her case the tapetoretinal degeneration was observed chiefly in the periphery of the fundus of each eye.

Consanguinity of the parents did not exist, but albinism, myopia and nystagmus were noted in several generations of the father's family. Bunge points out similarities of these two cases to those described by Krienes and by Birsch-Hirschfield, especially regarding the hemeralopia and the scarcity of the pigment in the fundus. In Bunge's opinion, his cases may be classified eventually in the group of those of tapetoretinal degeneration associated with amaurosis described by Leber.

K. L. Stoll.

Retinitis Pigmentosa. J. Bartók, Orvosképzés **24**: 625, 1934.

This paper begins with generalities on retinitis pigmentosa derived from the literature to date. The cardinal symptoms of this disease consist in clumps of pigment in the eyeground similar to bone corpuscles in addition to tubular restriction of the visual field and hemeralopia. The author ascribes to the disease twelve variations; the principal ones are supposed to be retinitis pigmentosa sine pigmento, retinitis punctata albescens, atrophia gyrata choroideae and choroideremia peripherica. The pathologic alterations in retinitis pigmentosa are, in the main, located in the neuro-epithelium and cause progressive primary degeneration. Histologic examination has shown that the choroid is not involved in the process. Retinitis pigmentosa is hereditary, its inheritance being of recessive or dominant character. It is transmissible to children of either sex from either the father or the mother. In from 25 to 40 per cent of the noted cases, consanguinity of the parents was manifest. Retinitis pigmentosa has thus far been attributed to very different causative agents. There is a supposed connection between retinitis pigmentosa and disease of the pituitary gland. Degeneration of the retinal pigment has been traced to disorders of the vegetative nervous system and, indirectly, to disturbances in the endocrine glandular system, chiefly of the parathyroid gland. Insufficiency of the renal function, too, is said to play a causative rôle. In many cases syphilis, especially hereditary syphilis, was regarded as an etiologic factor. Retinitis pigmentosa seldom occurs as an isolated disease; it is generally accompanied by some kind of maldevelopment of the eye. On the whole, two pathologic principal types of retinitis pigmentosa may be accepted, namely, the heredogenerative type and the endocrine type. Clinically, and especially ophthalmologically, it is, of course, impossible to discriminate between these two types. Until lately therapeutic measures were ineffective. At present ophthalmologists avail themselves of all-round curative procedures. No cure—not even improvement—can be obtained for the anatomic lesions of the morbid process; particular symptoms, according to the author, can be influenced beneficially.

N. Blatt.

Trachoma

TREATMENT OF TRACHOMATOUS CONJUNCTIVITIS IN CHILDREN. A. F. MACCALLAN, Rev. internat. du trachome **13**: 171 (Oct.) 1936.

The child of a mother with active trachoma can escape infection only by a miracle. Follicles on the upper tarsus are not pathognomonic of trachoma unless they are accompanied by infiltration and vascularization above the upper part of the cornea, or unless, as in the second stage, bleblike excrescences occur which rupture on pressure, with exudation of a gelatinous material. These excrescences must be destroyed by grattage at several séances. Conjunctival discharge is generally due to a superimposed bacterial infection, which necessitates instillation of antiseptic agents. Silver nitrate is of value, but the organic silver compounds are not.

J. E. LEBENSOHN.

LACRIMAL AND TRAUMATIC FACTORS IN UNILATERAL TRACHOMA. J. SÉDAN, Rev. internat. du trachome **13**: 184 (Oct.) 1936.

The evidence of unilateral trachoma varies according to the author and the region, the number of cases ranging from 0.56 to 12 per cent of all cases of trachoma. In a series of cases of this condition Sédan compared the lacrimal condition of the two eyes. The same proportion of impermeability occurred (in three of twenty-three cases), but spontaneous drainage of colored fluid into the nose was demonstrated on the healthy side in nineteen cases, as against five in which this was seen on the affected side. Traumatism also undoubtedly promotes the progress of trachoma, and caution must be exercised even in the use of ophthalmic ointments.

J. E. LEBENSOHN.

CLINICAL EXPERIENCES WITH LOCAL QUININE THERAPY IN TRACHOMA. H. C. CH'I, Chinese M. J. **50**: 1401 (Oct.) 1936.

Ten patients with trachoma at the ophthalmic clinic of the Peiping Union Medical College were treated with quinine bisulfate. Seven did not return to follow the treatment to completion, so only three could be followed for a considerable period.

A definite beneficial result was noted in each of the three cases. The patients were treated with a 10 per cent *solution* of quinine bisulfate, which was followed by only a moderate burning sensation. There was no damage to the cornea, even after *ointment* of this drug was applied twice a day over a long period. Ch'i realizes that a period of three months' observation is much too short for definite conclusions to be drawn.

S. H. McKEE.

Uvea

A CASE OF STRIATE CHOROIDITIS. A. MAY VIANNA, Ann. d'ocul. **173**: 954 (Dec.) 1936.

Striate choroiditis is an ophthalmoscopic picture rarely observed and seldom mentioned in textbooks. Details of a typical case are given, with a colored picture illustrating the appearance of the fundus in this condition.

Examination showed that the left papilla was slightly pale; the retinal vessels were normal, and the macular region was practically normal. The color of the fundus, which was more or less homogeneous, was lighter about the papilla. The condition was characterized particularly by a peculiar formation in the choroid of well defined yellowish orange streaks. These were beneath the retinal vessels, which crossed them at numerous points. In one region, at the temporal side above the disk, was seen one streak which ended by dividing into two branches. In general the choroidal lesions were considerably more marked toward the periphery than near the papilla.

Etiologically, this form of choroiditis, according to Wilmer, is related to tuberculosis, but in the case reported here tuberculosis could be excluded. The choroiditis began in the fourth month of pregnancy, which may have some bearing on the question of the etiology. The nature of this obscure form of chorioretinitis is interesting and may later be solved by histologic examination.

S. H. McKee.

Vitreous

Detachment of the Upper Portion of the Vitreous Body: Report of a Case. A. Pillat, Klin. Monatsbl. f. Augenh. **97:** 60 (July) 1936.

Pillat reports a case which enabled him to prove the existence of a detachment of the upper portion of the vitreous, a phenomenon which he had discussed theoretically some time previously. Complicated cataracts were extracted in the capsules from both eyes of a woman aged 61. Uneventful operations were followed by recovery, with vision of 6/9 in the right eye, and 6/6 in the left, with correction. The pupils were round.

The anterior part of the glassy membrane was observed to protrude into the anterior chamber of the right eye twenty-four days after the operation. The membrane was noticed to rest behind the pupil downward and nasally and within the pupillary plane temporally and above; its central portion protruded into the anterior chamber after dilatation of the pupil. A wavy line could be seen behind the glassy membrane. It was a tent-shaped detachment of the vitreous. A ring-shaped tear in the posterior part of the glassy membrane could be seen below and slightly nasal from the disk; it had an oval form from which three filiform prominences extended. A transparent and wavy membrane extended from the upper border of the tear to the tent-shaped detachment. Slit-lamp pictures and sketches of the fundus illustrate this condition. Pillat distinguishes three types of these detachments: the tent-shaped detachment, the funnel-shaped detachment of the upper portion of the vitreous and the detachment resembling a cul-de-sac, of the upper and posterior part of the vitreous.

The presence of such detachments explains some pathologic processes connected with detachment of the retina, which are discussed in detail.

K. L. Stoll.

Sympathetic Ophthalmia

PATHOGENESIS OF SYMPATHETIC OPHTHALMIA: REPORT OF A CASE OF SYMPATHETIC OPHTHALMIA CAUSED BY INTRA-OCULAR MELANOSARCOMA. W. H. MELANOWSKI, Klin. Monatsbl. f. Augenh. **97:** 52 (July) 1936.

Melanowski agrees with the authors who maintain that those cases of sympathetic ophthalmia due to melanosarcoma in which complete and typical symptoms are present are rare. He reports the case of a girl aged 12 which he considers typical. Vision of the left eye was gradually failing. A hemispherical detachment of the retina was observed. The Wassermann and Pirquet reactions were negative. Enucleation was refused. Iridectomy was done when inflammatory glaucoma developed in this eye, and sympathizing inflammation with involvement of the optic nerve in the hitherto healthy right eye set in a few weeks later, while hypertension persisted in the left eye. Histologic examination proved the presence of a melanosarcoma of the choroid after enucleation of the left eye; round and spindle-shaped cells were noted. The sympathizing right eye recovered slowly in the course of several months of treatment with intravenous injections of blood from persons with malaria, and with arsphenamine and quinine, and local therapy. No recurrence took place during five years' further observation, and vision was normal.

In this connection the case of another patient, aged 35, who died from metastases in several organs, is referred to. The melanosarcoma had spread through the chiasm into the second eye. Melanowski accepts the view of a number of authors that a chronic inflammation is the main secondary cause for the development of sympathetic ophthalmia in cases of an intra-ocular neoplasm and that tuberculosis may be an etiologic factor. An acute inflammatory process, such as angina or malaria, may produce recovery after removal of the offending eye. The second eye is involved by way of the blood vessels of the optic nerves.

K. L. STOLL.

Therapeutics

RADIOTHERAPY IN LESIONS ABOUT THE EYE. G. E. RICHARDS, Am. J. Roentgenol. **36:** 588 (Nov.) 1936.

Richards states that generally the eye tolerates radium rather well, and with proper precautions it may be used safely in a variety of conditions. The conditions in which it is dangerous are corneal ulcer, secondary glaucoma and cataract. Granular lids or eczema along the edges of the lids usually responds well to unfiltered roentgen radiation. Papillomas may be destroyed by single pole fulguration followed by a slight dose of radium. Simple rodent ulcer on the lid and remote from the lens or either canthus may be treated by a surface application of radium or by the implantation of highly filtered needles. The obvious dangers of treating lesions about the inner canthus are destruction of tissue resulting in contracture and deformity of the lids, and interference with the function of the lacrimal duct. In cases of lesions near the outer canthus with slight or no ulceration the treatment is carried

out by everting the lid, shielding the whole eye and the other tissues by a suitable lead shield and either applying radium on the surface or inserting highly filtered radium needles. The details of radium treatment are determined by the individual case but the treatment takes the form of a surface application, a pack applied externally or the implantation of highly filtered radium needles. In hyperplasia of the corneal epithelium, the eye is cocainized, and the lids are retracted and held in position by retractors. A tracing of the hyperplastic tissue is taken on transparent celluloid and transferred to a piece of lead foil, and an opening is cut in the latter, as in the treatment of lesions on the skin. A piece of rubber tissue 1 mm. thick is cut to fit over the entire cornea, extending down into each conjunctival sulcus, and is sterilized and placed in position. Over this and in direct contact with it is placed the lead foil shield, over the aperture of which the radium is applied. In Richards' cases the applicators were monel metal needles containing 10 mg. each of radium element, and these were left in contact for one hour. The treatment of an epithelioma developing on the cornea differs from the treatment of hyperplasia only in the matter of dosage, the malignant lesion requiring a heavier application. Richards has treated one hundred and two patients with malignant lesions of the lid, with radium with only three failures; all the other patients have been cured, so far as he has been able to follow them.

J. A. M. A. (W. ZENTMAYER.)

THERAPEUTIC LOWERING OF THE INTRA-OCULAR TENSION IN TABETIC ATROPHY OF THE OPTIC NERVE. J. SOBÁNSKI, Klin. Monatsbl. f. Augenh. **97:** 1 (July) 1936.

Tabetic atrophy of the optic nerve must be treated symptomatically and etiologically. The symptomatic treatment consists in an improvement of the circulation of the blood in the retina by reducing the intra-ocular pressure and by increasing the general blood pressure.

The etiologic treatment is antisyphilitic therapy. Strychnine and some other remedies cannot furnish good results because they raise the intra-ocular tension. The author reports thirty-seven cases to show that good results may be obtained by lowering the intra-ocular pressure considerably, even to below 10 mm. of mercury. Progress of the atrophy of the optic nerve could be prevented in some cases even without specific or tonic remedies, and vision, as well as the field of vision, could be improved, provided the process of atrophy had not arrived at the stage of stabilization. Sobánski calls this method *Entlastungstherapie,* as it has a decompressing (*entlasten*) effect on the circulation in the retina and in the intrabulbar portion of the optic nerve.

Thirteen of the thirty-seven patients had one blind eye. Improvement was obtained in forty eyes of thirty-one patients; no improvement of vision was noted in eleven eyes, and final deterioration occurred in ten eyes, four of which showed temporary improvement. The author refers to a previous publication for further detail (*Arch. f. Ophth.* **135:** 401, 1936). The duration of the treatment ranged from one year to twenty months.

K. L. STOLL.

DIAGNOSIS OF TUBERCULOUS DISEASES OF THE EYE AND THEIR TREATMENT WITH SUBCONJUNCTIVAL INJECTIONS OF VIRULENT HUMAN TUBERCLE BACILLI. J. KRASSÓ, Orvosképzés **25**: 267, 1935.

It is common knowledge that a disease of the eye may be of tuberculous origin even though the histologic examination shows no typical tuberculoma. With definite evidence of a tuberculous origin the involvement in question may display a nonspecific simple inflammatory reaction. In the etiologic diagnosis of tuberculosis of the eye one should make a diagnosis *ex juvantibus* in addition to bacillary and histologic investigations. A protein derived from virulent human tubercle bacilli (tebeprotin) is being used without causing any general or unpleasant focal reactions. The first therapeutic measure is in the form of conjunctival instillations. Later, two or three weeks after intradermal tests, subconjunctival injections of the tuberculin are given. These injections are made under the bulbar conjunctiva, without local anesthesia, by means of a very fine needle at a distance of 4 mm. from the sclerocorneal junction. Not until the conjunctival irritative phenomena have cleared up are the injections repeated. The dose of tuberculin to be injected is always measured in a relative sense, in that 0.03 cc. of the solution that gave a positive intradermal reaction is injected subconjunctivally. The subconjunctival injection is always followed by a conjunctival reaction. The reaction is designated as positive when it is coincident with a wide sector-shaped superficial dilatation of vessels and when a livid deep vascularization and tumefaction of the subconjunctival tissue are present. A strongly positive reaction manifests itself by the occurrence of episcleral nodules showing a simple lymphocytic infiltration of tuberculoid structure. Besides the positive reaction to the subconjunctival injection of the tuberculin therapeutic results are always present in the diseased eye. This, however, also happens frequently with a negative reaction. In none of the cases was the ocular disease aggravated as a consequence of the injections. It seems, according to Krassó, that local specific therapy with this tuberculin is yielding better results than subcutaneous specific therapy, and even better than climatotherapy.

N. BLATT.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Feb. 15, 1937

JOHN H. DUNNINGTON, M.D., *Chairman*

LEGRAND H. HARDY, M.D., *Secretary*

THE EYE OF THE EAGLE. DR. FREDERICK H. THEODORE.

The characteristics of the eye of the eagle are: marked convexity of the cornea; the presence of bone and cartilage in the sclera; the presence of striated muscle in the iris and ciliary body; an unusual structure of the lens; an avascular retina, which is extremely rich in cones and has two maculae, and a pecten, a vascular nutritive structure arising from the optic nerve and going anteriorly into the vitreous.

THE EMBRYOLOGIC DEVELOPMENT OF THE ANGLE OF THE IRIS. (Lantern Slides.) DR. A. L. KORNZWEIG.

The eyes of embryos ranging in age from 4½ weeks to 7 months were shown in cross-section, and the gradual development of the structures that go into the formation of the angle of the iris was shown. It was noted that these structures are all derived from the mesoderm. The relation of Descemet's endothelium to the trabeculum was pointed out, and the origin of Schlemm's canal was shown.

A METHOD OF PLOTTING BLINDSPOTS. DR. HENRY MINSKY.

A method of determining the blindspot was outlined, the scotomas being analyzed and differentiated by means of a photo-electric cell into: (1) absolute (determined centripetally); (2) relative (determined centrifugally), and (3) pseudopodial (determined by tangential and radial movements of the object).

ORBITAL TUMOR: A REPORT OF THREE CASES. DR. JOSEPH LEVINE.

The case histories of three patients were presented. One patient was a girl of 12 years from whose left orbit a lymphosarcoma was removed in 1934. Roentgen therapy was instituted, and there had been no recurrence of the growth, but a cataract developed in the left eye.

The second case was that of a woman 69 years old from whose left orbit a sarcoma was removed in December 1935. This sarcoma originated in plasma cells. Roentgen therapy was given in this case also, but no cataract developed.

The third case was that of a woman 28 years old from whose right orbit a simple fibroma was removed in January 1936.

It was emphasized that simple fibroma is extremely rare in the orbit.

DISCUSSION

DR. MARTIN COHEN: I should like to ask Dr. Levine whether the sections of the last specimen shown might be those of a neurofibroma, which is probably more common than fibroma. It appeared to me as though there might be some neural elements there, so that the tumor might be considered as a neurofibroma rather than a pure fibroma.

DR. JOSEPH LEVINE: There was no evidence of neurofibromatosis. The tumor was a fibroma.

TREATMENT OF OCCLUSION OF THE CENTRAL RETINAL ARTERY BY PARACENTESIS. DR. DAVID WEXLER.

A man aged 32, with mitral stenosis and occasional auricular fibrillation, had occlusion of the central retinal artery, which was undoubtedly embolic. Paracentesis was done within eight hours, and the anterior chamber was opened daily for one week. Acetylcholine and sodium nitrite were administered preoperatively, but were discontinued on the advice of the patient's physician. Vision did not improve until the fifth day, but was normal within ten days. The opinion was expressed that if vasodilators do not yield results within the first few hours following occlusion, paracentesis without further delay is indicated, especially in the absence of retinal arteriosclerosis.

PHARMACOLOGY OF HISTAMINE WITH REFERENCE TO THE EYE. DR. ROBERT K. LAMBERT and DR. HAROLD ABRAMSON.

The action of histamine on the eye was studied with a twofold purpose: 1. Because of the similarity of response to that encountered in allergic states, it was used as a simple means for investigating allergic conditions of the eye. 2. In order to determine practical applications, the pharmacologic effects of histamine were observed in some detail. Iminazolylethylamine and histidine produced no ocular responses.

DISCUSSION

DR. ROBERT K. LAMBERT: May I say one or two words to illustrate what we are driving at clinically? There is reason to believe that at least in some cases iritis is on a straight allergic basis. The whole reaction to histamine (the stimulation of the smooth muscles of the iris, the edema and the increased permeability of the blood vessels) may be considered analogous to the picture of an allergic iritis. One thing we are trying to do in the chemical approach is to find some way to offset the effect of histamine itself in the eye with various substances, in the hope that perhaps there will be a wedge by which the whole problem of allergy can be a little more satisfactorily attacked.

OCULAR MANIFESTATIONS OF NASOPHARYNGEAL TUMORS. DR. KAUFMAN SCHLIVEK.

This article is published in full in this issue of the ARCHIVES, page 1055. NOTES ON OPERATIVE PROCEDURES. (Lantern Slides.) DR. ISADORE GOLDSTEIN.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Feb. 18, 1937

C. R. HEED, M.D., *Chairman*

A. G. FEWELL, M.D., *Clerk*

DEVELOPMENTAL AND ANATOMIC FACTORS IN DACRYOCYSTITIS. DR. J. PARSONS SCHAEFFER.

The origin and development of the efferent lacrimal passageways not only clarify the general plan of the structure of these passageways but the anatomic types and anomalies as well. From the deep layer of the ectoderm along the line of the obliterated naso-optic groove grows a solid strand or cord of epithelial cells, the rudiment or anlage of the nasolacrimal passageways. This cord of epithelial cells becomes detached from the surface and surrounded by mesenchyme cells. Sprouting occurs at both the nasal and the ocular end, thereby connecting the solid epithelial cord with the free borders of the eyelids and the mucous membrane of the inferior nasal meatus.

A lumen forms within the solid cord, which begins at the ocular end of the parent cord and progresses toward the nasal end, being well advanced in development in embryos of 100 days. The horizontal portions of the lacrimal ducts or canaliculi become canalized before the vertical portions. Canalization of the areas of union between the free borders of the eyelids and the lacrimal ducts is often deferred until a later period and occasionally does not take place at all, variously resulting in atresia at these points. Frequently at birth the lacrimonasal membrane between the nasal end of the nasolacrimal duct and the inferior nasal meatus remains unruptured, giving rise to atresia at the usual point of outlet. The membrane may become sufficiently attenuated during the first months of infancy to rupture spontaneously. If the aperture does not become established, operative intervention is required.

Normally canalization of the solid nasolacrimal rudiment occurs irregularly, and at an early time there is abundant evidence of mucosal ledges, valves and diverticula. Secondary buds from the stem rudiment account for the larger diverticula which are encountered in later life. The belief that these diverticula are acquired in adult life as the result of disease is not justified. They have an embryonic origin.

From the genetic point of view, the variations in the structure of the adult nasolacrimal passageways fall into three important categories or groups: first, those that have their origin in rudiment potentials; second, those due to early arrest in the development of parts which should proceed from the stem rudiment, and, third, those due to division of previously continuous ducts into discontinuous segments, the latter probably caused by amniotic bands. Rudiment potentials explain such variations and abnormalities as supernumerary lacrimal ducts of one or both eyelids; duplication, triplication, etc., of the lacrimal puncta; the solid and furrow-like lacrimal punctum; mucosal ledges and so-called valves within the nasolacrimal duct, irregularities of contour and diverticula; variation in the structure of the lacrimonasal aperture in

the inferior nasal meatus, and osseous and membranous segments of the main nasolacrimal duct. Developmental arrest leads to other abnormalities.

The plane of direction of the osseous nasolacrimal canal conforms to the type of facial skeleton. The breadth of the bridge of the nose, the width of the inferior nasal meatus and the degree of expansion of the piriform aperture influence the course or direction of the osseous nasolacrimal canal. Generally speaking, one may say that the osseous nasolacrimal canal is projected caudad, laterad and dorsad along a plane erected from the lacrimal fossa to the interval between the second premolar and the first molar or the first and the second molar, or even as far dorsad as the interval between the second and the third molar. The almost vertical plane frequently shown in textbooks is erroneous.

An important anatomic feature of the mucous membrane of the lacrimal sac is the presence beneath the epithelium of a relatively thick fibro-elastic tunica propria with an abundance of lymphoid tissue, some of which is arranged in definite masses not unlike the aggregated nodules of the small intestine. Small tubular glands are found, and frequently numerous cells exhibit stages of conversion into mucus-containing goblet cells. Another anatomic feature is the loose tissue which connects the tunica propria with the periosteum. This tissue everywhere is occupied by a rich plexus of veins which partakes of the nature of a modified erectile tissue. The blood spaces, which are under control of the autonomic nervous system, are large, and the stratum which contains them is subject to rapid engorgement and depletion. The extensive venous plexus of the nasolacrimal sac and the nasolacrimal duct is believed to be a factor in the flow of tears from the conjunctival cul-de-sac to the inferior nasal meatus.

As early as the eighteenth month of infancy the nasolacrimal duct bears an intimate relationship to the maxillary sinus. The ethmoid cells which develop from the frontal recess and from the ethmoid infundibulum are nearest the lacrimal sac at birth. The topographic relationship is, however, not intimate at this time. By the fifth or the eighth year the anatomic relationships between the paranasal sinuses and the lacrimal fossa are, in essence, those of the adult. In the latter, the frontal and maxillary sinuses and the anterior group of ethmoid cells need to be considered in this connection, and of these the anterior ethmoid cells are of special importance. Indeed, at times the walls of the lacrimal fossa and the upper part of the osseous nasolacrimal canal are pneumatized by ethmoid cells variously related to the lacrimal sac and the upper part of the membranous nasolacrimal duct.

From a clinical point of view the lacrimonasal aperture or ostium in the inferior nasal meatus may be of one of five types: (1) the type which fails to canalize (that is, the lacrimonasal membrane remains intact); (2) that of microscopic size and wholly inadequate for the function intended; (3) that located at the highest point of the inferior nasal meatus, with osseous walls, wide mouthed, permanently open and unguarded by any valvelike fold of mucous membrane; (4) that located in the mucous membrane of the lateral wall of the inferior nasal meatus some distance below the attached border of the inferior nasal concha and frequently guarded by a valvelike structure of mucous membrane;

(5) that located in the mucous membrane of the lateral wall of the inferior nasal meatus without an adequate valve from the point of view of physiology. The anatomic type of lacrimonasal aperture is undoubtedly a factor in satisfactory drainage of tears into the inferior nasal meatus and a regurgitant infection of the lacrimal sac in nose blowing.

The origin and development of the efferent lacrimal passageways point the way to a better understanding of the congenital variations and defects. A knowledge of the developmental history and potentialities is a definite aid in deciding on the course of treatment of the several types of congenitally atypical ducts. The important anatomic types of departures from the so-called typical passageways have a bearing in disease, prognosis and treatment. Although in many nasolacrimal ducts the lacrimal probe can be successfully passed, there are anatomic types in which probing cannot be performed with any degree of success, and much injury may result. In the latter cases conservative treatment would be much more effective than probing.

It is to be hoped that roentgenographic study will be extended to include the lacrimal passageways in the living body in order that the rhinologist may gain some knowledge of the anatomic character of the nasolacrimal passageways which he is about to treat.

DACRYOCYSTORHINOSTOMY. DR. ALVIN HOWLAND.

This article is a survey of a large number of cases in which patients were treated by external dacryocystorhinostomy. The results obtained are compared with those in a similar number of cases in which patients were treated by the ordinary removal of the lacrimal sac.

DISCUSSION

DR. EDMUND B. SPAETH: There are some points which must be emphasized, in Dr. Howland's paper, relative to indications and contraindications for dacryocystorhinostomy. Also, I wish to say something about the complications and the reasons for the failures which occur. I feel that most of these failures are unnecessary. First, relative to the bony opening, too large an osteotomy opening will close in so that the retained portion of the sac will be too small to permit bridging of the bony opening. In such instances healing cannot occur between the nasal mucosa and the periosteum at the edges of the osteotomy opening with the edges of the retained portion of the sac. Second, tearing the internal common punctum while probing during the operation is a cause for failure. Third, too extensive resection of the wall of the sac itself is also a cause for failure. The mechanics is rather similar to that in cases in which too large an osteotomy opening has been made. If the resection of the wall of the sac is more extensive than it should be, the sutures which must be inserted from the periosteal edges of the osteotomy opening to the margin of the sac itself will drag the sac out of the osteotomy opening as a taut diaphragm. The bony opening will then close beneath this, and as a result one has neither a dacryocystorhinostomy nor a dacryocystectomy. Fourth, failure to resect the bone to a proper level will permit a lake of pus to remain undrained at the superior extremity of the bony lacrimal nasal

duct. The structure of the bony fossa varies to a great degree, and the bone must be resected to a level equal to the lowest level of the sac regardless of whether one is working with the lacrimal bone or at the lacrimal crest of the malar bone. Fifth, adequate postoperative intranasal attention is necessary; that is, the nose must be kept free from clots and crusts by irrigation through the punctum and by the use of a combination of liquid petrolatum and ephedrine.

In regard to the contraindications, chronic ethmoiditis is not necessarily a contraindication unless polypi are present. If these are present they must be cleared up first. The same principle applies to an enlarged tip of the middle turbinate bone. If it will not shrink out of the way, a partial turbinectomy must be done. Traumatic obstructive dacryocystitis responds well to a dacryocystorhinostomy, though there is no doubt that it is easier to operate in cases of chronic suppurative dacryocystitis in which there is a large dilated cystocele. Intra-ocular surgical intervention can be done, and has been done, without the fear of complications after a successful dacryocystorhinostomy. Fistulas, when present, heal spontaneously and promptly after a rhinostomy. Additional surgical intervention is not necessary because of these. Chronic atrophic rhinitis is a rather definite contraindication. Osteomyelitis, if present, is likely to be tuberculous. This is a contraindication. A dacryocystorhinostomy in the presence of slit canaliculi will not correct the epiphora. If these cannot be bridged over by the repeated application of cautery, one should proceed with a cystectomy.

Occasionally during the operation one may open into an anterior ethmoid cell. The osteotomy should be continued as if this complication had not occurred. It is wise, however, to scrape with an ethmoid curet the anterior ethmoid cells which were opened so that the osteotomy opening will not close as a result of suppuration due to infection of an ethmoid cell. This has happened in several instances as a result of suppuration due to infection of an ethmoid cell, and in each instance the patient was referred to a rhinologist for subsequent treatment of the ethmoid cell. There were no failures as a result of this complication.

DR. FRANCIS HEED ADLER: Is there any danger in accidentally perforating an anterior ethmoid air cell by making the window in the bone too high up?

The most frequent objection which is heard against this operation is that it is not suitable for a patient on whom an operation for cataract is contemplated, on the ground that infection can spread directly from the nose into the conjunctival cul-de-sac. The advocates of the operation maintain that there is no more danger of infection than after a dacryocystectomy. They reason, and perhaps rightly so, that the flow of tears into the nose prevents ascending infection, whereas in the old operation of dacryocystectomy bacteria were able to multiply in the conjunctival cul-de-sac owing to stagnation of the lacrimal fluid. It would be interesting to compare cultures from the conjunctival cul-de-sacs of a series of patients on whom a dacryocystectomy had been done on one side and a dacryocystorhinostomy had been done on the other. I think this would give a truthful answer to the question.

DR. ALVIN HOWLAND: In the number of cases I have observed I have never seen one in which the ethmoid cells were exposed. The periosteum would act as a protection in case any of them should be

exposed, on account of the elevation, and they would then fall back into place. On several of the patients in this series my associates and I later did extraction of cataract. All showed an uneventful recovery.

CONGENITAL IMPERFORATION OF THE NASAL LACRIMAL DUCT. DR. WILLIAM ZENTMAYER.

The term dacryocystitis is considered inappropriate for congenital imperforation of the nasal lacrimal duct. Several possible causes are enumerated, but failure of absorption of the epithelial cord, with imperforation at the meatal opening, is considered the most common. The two principal complications are infection of the epithelial contents of the sac, producing true dacryocystitis and at times abscess.

The symptomatology is described, and the differential diagnosis between this condition and ophthalmia neonatorum and various forms of prelacrimal cyst is gone into. The prognosis is considered good if the obstruction is removed early. Probing is strongly urged to prevent infection and abscess and the possibility of infection in case of a corneal injury or ulcer and also because of the satisfaction of the parents in having the blemish removed. In only two instances in my experience was more than one probing required. The procedure can be carried out without general anesthesia, as the probe meets with no firm obstruction in its passage.

ROYAL SOCIETY OF MEDICINE, LONDON, SECTION OF OPHTHALMOLOGY

Clinical Meeting, London, England, March 12, 1937

MR. W. H. MCMULLEN, O.B.E., F.R.C.S., *President*

TRANSITORY TREMULOUS LENS. DR. M. E. ALVARO, SÃO PAULO, BRAZIL.

A man aged 29 when first seen had uveitis in his left eye. In two previous attacks the condition was regarded and treated as syphilitic, though the Wassermann reaction was negative. There was a good deal of exudate in the aqueous and the vitreous of the right eye, which showed slight hypotension and vision adequate to counting fingers at 3 feet (91.4 cm.). Several foci of dental sepsis were revealed by the roentgen rays, but no evidence of tuberculosis or other septic foci was discovered. The dental foci were removed, and after six injections of foreign protein both the aqueous and the vitreous began to clear, and vision gradually improved to 6/60. Seven weeks after the first examination, however, the condition suddenly recurred; vision was mere perception of light, and both the aqueous and the vitreous were almost opaque with exudates. At that time iridodonesis and tremor of the lens could easily be seen, the tremor increasing during the next few days, thereafter gradually decreasing, and disappearing entirely nine weeks after it was first seen. Vision gradually improved to 6/36. Five weeks later the vision of the eye decreased suddenly, and a large

retinal detachment was found. Šafář's operation was carried out, with good results. Seven months after the operation the retina remained completely reattached, and vision had improved to 6/12. The fluidity of the vitreous might have caused the lens to be tremulous, as D'Ombrain has suggested. In his case and in this case the intra-ocular tension was lower than normal.

CONGRUOUS FIELD DEFECTS DUE TO CONGENITAL ABSENCE OF NERVE FIBERS. MR. P. G. DOYNE.

The absence of the upper central part of the field of vision was only recently noticed in an infant aged 1 year. Vision of the right eye was 6/36, not improved; vision of the left eye was 6/6. There were practically congruous field defects extending from the optic disks over the upper nasal and part of the upper temporal quadrants. There was no evidence of previous intra-ocular disease. There were symmetrical pits in the lower part of each optic disk, associated with proliferation of pigment. Neurologic examination revealed no abnormalities.

DISCUSSION

MR. W. H. MCMULLEN: The difficulty is to account for the association in the same patient of congruous field defects with the symmetrical abnormalities of the disk.

MR. EUGENE WOLFF: Mr. Hyne suggested to me that the condition of this patient might be similar to that of a patient whom I showed before the Ophthalmological Society of the United Kingdom at its meeting in Edinburgh in 1931. I suggested at that meeting that the most common cause of recurrent proptosis was lymphangioma of the orbit, or, as it is sometimes called, cystic hygroma of the orbit, characterized by the fact that the patient is liable to recurrent attacks of inflammation. Possibly this may be a case of cystic lymphangioma, as in that condition the proptosis may come on in a few hours.

RETINAL CHANGES IN A CASE OF PURPURA. MR. P. G. DOYNE.

A girl aged 10 years had fits of the jacksonian type three months before examination. The condition was thought to be due to subdural hemorrhage. There was no gross anemia. Temporary improvement took place after transfusions of blood. During treatment subconjunctival hemorrhages occurred bilaterally. On ophthalmoscopic examination retinal hemorrhages were found in each eye. Splenectomy was performed, and four days later the platelet count had enormously improved, namely, to 318,000 per cubic millimeter. On discharge considerable absorption of the hemorrhages had taken place, and vision of the left eye was 6/12, and that of the right, 6/36. There were small radial hemorrhages overlaid by white exudate; the veins were slightly engorged. The present vision is 6/9 in the right eye and 6/12 in the left eye.

A CASE OF UVEOPAROTITIS. MR. P. G. DOYNE.

In a patient with uveoparotid inflammation, facial paralysis was the first feature noted, and there was no sign of tuberculosis in the chest or in any other part of the body.

DISCUSSION

MR. CHARLES GOULDEN: I thought it was established that this condition was due to tuberculosis. In comparatively recent times I observed two examples of it, and the patients were in the ward at the same time. The condition was severe, the corneas being smeared over with large masses of exudate. Both patients were treated as if they were tuberculous, and they made astonishing recoveries.

CHOROIDAL GROWTH. MR. T. E. DAVIES.

A slightly raised black mass at the outer side of the macula was found in a woman who came for refraction and who had visual acuity of 6/6 with each eye. The mass was too much raised to be a subchoroidal hemorrhage. The iris was clear. The change in the last month has been an increase in the size of the growth, and there has been some macular edema during the past few weeks. The Wassermann reaction was negative.

DISCUSSION

MR. CHARLES GOULDEN: This mass is apparently walled, and there are no changes in the vitreous. The blood vessels have passed through this mass. I do not know what this can be except sarcoma.

MR. W. H. MCMULLEN: I agree that this growth is probably sarcoma, and I am convinced that one is not justified in allowing the patient to retain the eye.

GROWTH OF THE LACRIMAL SAC. MR. G. G. PENMAN.

A case is presented in order to elicit opinions about the nature of a growth of the lacrimal sac, especially to determine whether it is a granulating papilloma, tending toward malignancy.

DISCUSSION

MR. EUGENE WOLFF: I took sections of this mass, and six pathologists gave six different opinions concerning it. After looking at it carefully I concluded that it was papilloma, and at points where it was cut transversely across it gave different appearances. At no place is the epithelium growing into the deeper tissues, thus making it definitely malignant, but the wall of the central area of blood vessels is proliferating in part, and so I think it is on the borderline of malignancy. I believe that local removal would cure the condition.

SARCOMA OF THE CHOROID; RETINITIS EXUDATIVA EXTERNA. MR. EUGENE WOLFF.

A man, first seen at the Royal Westminster Ophthalmic Hospital on Oct. 28, 1936, complained of mistiness of the left eye of three weeks' duration. The right eye was normal, with vision of 6/9 partly. The left eye had vision of 6/6 partly, and the left fundus showed extensive subhyaloid hemorrhage in the upper and nasal portions. The Wassermann reaction and the results of general physical examination were negative. The hemorrhage was absorbed, leaving a raised whitish mass at its site, the edges rising sharply from the surrounding retina. The

retinal vessels disappeared beneath the mass, to reappear above and nasally. Transillumination gave negative results.

This case may be similar to those cases of choroidal sarcoma described by Professor Rønne in the 1936 *Transactions of the Ophthalmological Society of the United Kingdom*. These were characterized by the fact that the patient when first examined had a vitreous hemorrhage which obscured the details of the fundus, and only later, when this cleared up, was the tumor seen. The hemorrhage tears through the retina so that the growth fungates freely through into the vitreous and explains how the retinal vessels may run deep to the growth. My associates and I cannot exclude a mass formed by changes in the blood clot, and at the moment the case is being observed.

DISCUSSION

MR. BASIL GRAVES: Some years ago a patient came to the Moorfields Hospital who had a small growth arising from the stroma of the iris, and the patient said that about every quarter, for four years, she saw red. A hemorrhage happened to start while I was making the examination. The stroma was transluminous, and a hole could be detected in the stroma of the iris by this means, but by no other.

Book Reviews

Controlled Reading; A Correlation of Diagnostic, Teaching, and Corrective Techniques. By Earl A. Taylor. Price, $3.50. Pp. 367, with approximately 100 plates. Chicago: the University of Chicago Press, 1937.

This is the first attempt to bring together in one compact volume the scattered results of many workers in the field of developing an efficient reading technic. Given perfectly normal eyes, one can still be an inefficient reader because of wrong or, rather, inefficient habits, for instance, the habit to linger over words or phrases after their content has been mentally noted, or the converse, the habit of reading too fast for comprehension and turning back for rereading. Every one will agree with Taylor's first premise that efficient reading habits are of paramount importance in childhood and youth, i. e., during the period of life devoted entirely to obtaining an education. Efficient reading is, of course, also of great importance in adult life, since the efficient use of the eyes for work or pleasure contributes toward a fuller and richer life.

The book is divided into four parts. Part 1 and a section of part 2 deal with the historical background of the subject. In part 1 a brief account is given of the investigators who have contributed to the science of efficient reading. As one would expect, the great majority of these were psychologists, teachers and educators in general.

However, though the educators have done by far most of the investigating, priority for attacking this problem goes to that eminently versatile ophthalmologist Louis Emile Javal. In 1878 he published the first article on this subject in the *Bulletin de la Société de médicine.* Next comes the work of the venerable educator and editor Dr. James McKeen Cattell, who in 1885 first published the results of an investigation into the time it takes to recognize and name letters, pictures and colors. From then on the stream of study and investigation widened, slowly at first and then more and more rapidly, until now there is an extensive and varied literature on the subject scattered through numerous publications.

The first section of part 2 describes the development of devices for measuring objectively the extent, number and rapidity of movements of the eye in reading, from the earliest crude methods by direct observation through the methods of mechanical and electrical recording to what seems to be the most comprehensive method, that of photography. Various models of binocular cameras for photographing movements of the eye are described. The detailed descriptions and photographs of the mechanics of the various models are probably of little interest to the physician, though they may be to some other readers. For the book is written for a varied group of readers.

However, starting with chapter IV of part 2, the ophthalmologist will find matter of real interest to him. A study of binocular movements of the eyes as recorded by the binocular camera has yielded some almost startling results. For instance, it was found that in many cases, though the eyes have what appears to be single binocular vision, yet "the eyes do not move together necessarily in the act of reading." In fact, with some readers "the rate of movement is not the same in the two eyes

and they do not move in the same direction." Taylor tries to interpret these findings in terms of "fusional conflicts" and "muscular irregularities." Possibly this interpretation is correct; possibly also the entire present conception of a geometric point to point correspondence in binocular vision (even including the extension of "Panum's areas") has to be revised. The studies and graphs in this book show that fixation in general is not as absolute as has heretofore been believed, nor is binocular fixation so mechanically accurate as the straight line diagrams illustrating binocular fixation would lead one to believe. Taylor's contention that a "binocular reading graph is an essential part of any comprehensive eye examination" is open to question, though such a graph is of interest and value. The statement that a reading graph is valuable because it gives "objective information concerning the maturity of the reading habit, or the functional efficiency of the subject in the reading situation" is probably a more conservative and more acceptable evaluation of the reading graph.

Part III discusses the remedial measures for inefficient reading. An instrument designed for this purpose, the metronoscope, is described, and the methods of using it for individuals and for classes are discussed and illustrated. The purpose is to "increase the reading rate without decreasing comprehension." It is unquestionably true that almost every one can learn to read more rapidly by simply willing to do so and applying this will to ordinary reading. For children and some adults, however, the use of a special instrument with objective control and measure of progress is helpful and sometimes indispensable. However, one must not overlook the fact that some printed matter needs only a superficial hasty reading, some a more leisurely reading and some a slow and protracted reading. The philosophic treatises of Kant or Spinoza must be read slowly because of the thought processes necessary for full comprehension of the context. One must guard against the speed mania of modern life entering too sharply into the field of reading. Taylor does mention comprehension of the matter read as an essential in effective reading, but the emphasis on rapidity is apt to overshadow the greater importance of comprehension and retention.

Part IV presents the results of an experimental study on the relation between various ocular defects and reading efficiency. A comparative study was made with a group of normal students and a group of failing students, called, respectively, "normals" and "failures." Under "ocular defects," refractive errors, muscular imbalances and visual suppression were considered. The results are expressed in several tables. Some of the findings are entirely in accord with what one would expect; e. g., of the failing students, only 3 per cent wore glasses, though 31 per cent needed them, whereas of the normal students, 15.50 per cent wore glasses of 23.51 per cent who needed them. Some other findings are a bit surprising, e. g., the fact that the failing students showed better stereopsis than the normal students, that there seemed to be "little relationship between visual acuity as an isolated factor and reading efficiency," and that "near phoria probably cannot be considered an important factor in diagnosing reading disability." According to the experiments, refractive errors, suppression and insufficiency of convergence are especially associated with ocular discomfort in reading. In general, it may be said that some of the results obtained from these experiments will not square with the experience of many ophthalmologists. Possibly the

manner of making the various visual, refractive and muscular tests in the series of experiments is partly responsible.

There is a further series of graphs to show the relation of visual efficiency to the successful study of mathematics. Here the author is both cautious and bold. For instance, the statement that from the data presented "there is a foundation for the belief that visual inefficiency influences the success of the student of mathematics" is too guarded. One would accept such a belief a priori. But he is rather bold immediately afterward when, in stressing the beneficial effects of exercises in prism reading, he says of a group of students who were failing in mathematics, "In almost all these cases it is probable that prism-reading exercises, or lenses, or both would have produced comfortable vision and in a few instances doubtless the need for lenses would have been eliminated by these exercises." Ophthalmologists, while according value to exercises in prism reading, which are essentially exercises in adduction and abduction, will probably question that statement.

The ophthalmologist will have occasion to question other findings as presented. It seems that much of the experimental work, so far as it involves a determination of the refractive, visual and musclar anomalies, was not done by experienced ophthalmic specialists. Psychologists, teachers and educators, with all due respect to their efficiency in their accustomed lines of work, have not the background of experience for fully evaluating the tests made for visual, refractive and muscular anomalies.

The last four chapters of part IV give directions and methods for using the metronoscope for individuals and for classes, for diagnosing inefficient reading habits and for correcting them. A great deal of stress is laid on the use of prisms base in, out, up and down, with or without auxiliary plus and minus lenses for practice with the metronoscope. Many successful results are cited. But, possibly for the reasons already mentioned, some of these seem a bit "raw" to the ophthalmologist. For example, a case is reported in which a 15 year old girl with a divergent strabismus of 12 prism diopters in the right eye, of eight years' standing, had visual acuity of 20/50 in that eye, with and without correction. Visual acuity of 20/30 developed in nine weeks, and, three months later, visual acuity of 20/20. The latter is what one sometimes experiences. But it is further stated that after nine weeks the patient could accept 30 prism diopters base out and after three months more could accept 60 prism diopters base out without diplopia. It is difficult to understand how a patient with normal vision in both eyes whose fusion power in adduction was so strong even after the lapse of eight years of nonuse could have had a divergent squint in the first place.

There is an extended bibliography, which will enable one interested in any phase of the work to pursue it further. There is also a complete index. The mechanical setup of the book—the print, the plates, etc.—is excellent.

It is understandable that the enthusiasm of an author who is working in a more or less new field is apt to carry him too far in one direction. This, however, is a minor fault if the reader is at all critical. To the ophthalmic specialist the book, in spite of a few questionable interpretations, presents a wealth of information which will prove of definite value to him.

JOSEPH I. PASCAL.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.
President: Dr. P. Bailliart, 66, Boulevard Saint-Michel, Paris (6e).
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the President, Dr. P. Bailliart.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Costzeedijk 316, Rotterdam, Holland.
Place: Cairo. Time: Dec. 8-14, 1937.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33, Welbeck St., London, W., England.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. P. E. H. Adams, 6, Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13, Mitchell Ave., Jesmond, Newcastle-on-Tyne.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.

Midland Ophthalmological Society

President: Dr. T. E. Ashdown Carr, 34, Charnwood St., Derby, England.
Secretary: T. Harrison Butler, 81, Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.
Time: Oct. 1, 1937.

Ophthalmological Society of the United Kingdom

President: Mr. Gordon M. Holmes, 9, Wimpole St., London, W. 1.
Secretary: Mr. J. D. M. Cardell, 27, Weymouth St., London, W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 8-10, 1937.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warszawa.
Place: Lindley'a 4, Warszawa.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.
Secretary: Dr. A. Rugg-Gunn, 35, Harley St., London, W. 1.
Time: June 11, 1937.

SOCIÉTÉ FRANÇAISE D'OPHTHALMOLOGIE

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. F. Berg, Uppsala, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. William L. Benedict, 102 Second Ave., S. W., Rochester, Minn.
Secretary: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Place: Atlantic City. Time: June 7-11, 1937.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Frank E. Burch, 408 Peter St., St. Paul.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Palmer House, Chicago. Time: Oct. 10-15, 1937.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.
Place: Hot Springs, Va. Time: June 3-5, 1937.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Road, Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. A. J. Ridges, Walker Bldg., Salt Lake City, Utah.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Salt Lake City, Utah. Time: May 24-27, 1937.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. H. Klemptner, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, Virginia Mason Hospital, Seattle.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. A. H. Pember, 500 W. Milwaukee St., Janesville, Wis.
Secretary-Treasurer: Dr. W. H. Elmer, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. L. H. Hohf, Yankton, S. D.
Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Secretary: Dr. O. M. Marchman, Medical Arts Bldg., Dallas, Texas.

Southwestern Michigan Triological Society

President: Dr. O. B. McGillicuddy, 1908 Capitol Band Tower, Lansing, Mich.
Secretary-Treasurer: Dr. Maurice C. Loree, 120 W. Hillsdale St., Lansing, Mich.
Time: Third Thursday of alternate months.

Western Pennsylvania Eye, Ear, Nose and Throat Society

President: Dr. C. W. Beals, Weber Bldg., DuBois.
Secretary-Treasurer: Dr. C. W. Beals, Weber Bldg., DuBois.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 227 16th St., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Walter L. Hogan, 750 Main St., Hartford.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.
Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Building, Atlanta, Ga.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. R. Dillinger, French Lick.
Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. James A. Downing, 406 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Des Moines.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. C. A. McWilliams, G. and S. I. R. R. Bldg., Gulfport, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Secretary-Treasurer: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. C. Coulter Charlton, 124 S. Illinois Ave., Atlantic City.
Secretary: Dr. H. L. Harley, 124 S. Indiana Ave., Atlantic City.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.
Secretary: Dr. Marvin F. Jones, 121 E. 60th St., New York City.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Trygve Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 514 6th St., Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary-Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. E. Houston, 103 E. North St., Greenville.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. W. Potter, 601 Walnut St., Knoxville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1937.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Edwin W. Burton, University of Virginia, University.
Secretary-Treasurer: Dr. George G. Hankins, 202 Medical Arts Bldg., Newport News.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Secretary: Dr. J. E. Blaydes, First National Bank, Bluefield.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Samuel T. Hubbard, 294 State St., Hackensack, N. J.
Secretary: Dr. William F. McKim, 488 Sanford Ave., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, First-Central Tower, Akron.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Jesse W. Downey Jr., 529 N. Charles St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Thurber LeWin, 112 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. G. Henry Mundt, 30 N. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.
Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.
Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Andrew Timberman, 21 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Claude S. Perry, 40 S. Third St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. A. W. Davidson, City National Bank Bldg., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 720 Medical-Professional Bldg., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Hugh L. McLaurin, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. A. W. Greene, 148 Barrett St., Schenectady.
Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 500 Metz Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Henry C. Haden, 1914 Travis St., Houston, Texas.
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. C. Daniel, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Davis, 1101 Grand Ave., Kansas City, Mo.
Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. Isaac H. Jones, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.
Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Hychener, 130 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. A. G. Farmer, 1040 Fidelity Bldg., Dayton, O.
Secretary-Treasurer: Dr. Rome M. Webster, 663 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., West, Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Secretary-Treasurer: Dr. Fowler Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. John H. Dunnington, 30 W. 59th St., New York.
Secretary: Dr. LeGrand H. Hardy, 30 E. 40th St. New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. W. H. Stokes, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. John S. Van Winkle, 297 Broadway, Paterson, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Falk Clinic. Time: 4 p. m., second Friday of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. A. F. Bagby, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Carl T. Eber, 308 N. 6th St., St. Louis.
Secretary: Dr. W. M. James, 508 N. Grand Ave., St. Louis.
Place: Oscar Johnson Institute Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. John T. Crebbin, 624 Travis St., Shreveport, La.
Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. A. Veasey Jr., 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Philip B. Green, Old National Bank Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. J. Werfelman Jr., 725 State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse. N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W. Toronto.
Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. James M. Greear Jr., 1740 M St., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Ernest Sheppard, 927 17th St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.

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